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=> b medline caplus lifesci embase uspatfull biosis
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                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

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=> s haloethane and pain
L1            2 HALOETHANE AND PAIN

=> s halothane
L2        67441 HALOTHANE

=> s l2 and pain
L3         2608 L2 AND PAIN

=> s l3 and intrathecal?
L4          475 L3 AND INTRATHECAL?

=> s l2 and psd93
L5            2 L2 AND PSD93

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6            2 DUP REM L5 (0 DUPLICATES REMOVED)

=> d l6 ibib abs tot

L6   ANSWER 1 OF 2  USPATFULL
ACCESSION NUMBER:        2002:85548  USPATFULL
TITLE:                   Inhibition of interaction of   ***PSD93***   and PSD95
                         with nNOS and NMDA receptors
INVENTOR(S):             Tao, Yuanxiang, Baltimore, MD, UNITED STATES
                         Johns, Roger A., Reistertown, MD, UNITED STATES

                              NUMBER         KIND    DATE
                         --------------- ------ --------
PATENT INFORMATION:      US 2002045590      A1   20020418
APPLICATION INFO.:       US 2001-853895     A1   20010514  (9)

                                NUMBER         DATE
                         ------------------ --------
PRIORITY INFORMATION:    US 2000-242580P    20001023 (60)
DOCUMENT TYPE:           Utility
FILE SEGMENT:            APPLICATION
LEGAL REPRESENTATIVE:    BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100,
                         WASHINGTON, DC, 20001
NUMBER OF CLAIMS:        65
EXEMPLARY CLAIM:         1
NUMBER OF DRAWINGS:      4 Drawing Page(s)
LINE COUNT:              1513
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB      PSD-95/SAP90 antisense-treated animals not only experience a significant
        decrease in MAC for isoflurane, but also experience an attenuation in
        the NMDA-induced increase in isoflurane MAC. PSD-95/SAP90 appears to
        mediate the role of the NMDA receptor in determining the MAC of
        inhalational anesthetics. Suppression of the expression of PSD-95/SAP90
```

mediated through the N-methyl-D-aspartate receptor activation. In spinal cord neurons PSD-95/SAP90 interacts with the N-methyl-D-aspartate receptor subunits 2A/2B. Activation of the N-methyl-D-aspartate receptor in spinal hyperalgesia results in association of the N-methyl-D-aspartate receptor with PSD-95/SAP90. PSD-95/SAP90 is required for hyperalgesia triggered via the N-methyl-D-aspartate receptor at the spinal cord level.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:850924 CAPLUS
DOCUMENT NUMBER: 135:366767
TITLE: Inhibition of interaction of ***psd93*** and psd95 with neuronal nitric oxide synthase and NMDA receptors
INVENTOR(S): Johns, Roger A.; Tao, Yuanxiang
PATENT ASSIGNEE(S): The Johns Hopkins University, USA
SOURCE: PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001087285	A2	20011122	WO 2001-US15372	20010514
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2002045590	A1	20020418	US 2001-853895	20010514
PRIORITY APPLN. INFO.:			US 2000-203894P P	20000512
			US 2000-242580P P	20001023

AB PSD-95/SAP90 antisense-treated animals not only experience a significant decrease in min. alveolar concn. (MAC) for isoflurane, but also experience an attenuation in the NMDA-induced increase in isoflurane MAC. PSD-95/SAP90 appears to mediate the role of the NMDA receptor in detg. the MAC of inhalational anesthetics. Suppression of the expression of PSD-95/SAP90 in the spinal cord significantly attenuates responses to painful stimuli mediated through the N-methyl-D-aspartate receptor activation. In spinal cord neurons PSD-95/SAP90 interacts with the N-methyl-D-aspartate receptor subunits 2A/2B. Activation of the N-methyl-D-aspartate receptor in spinal hyperalgesia results in assocn. of the N-methyl-D-aspartate receptor with PSD-95/SAP90. PSD-95/SAP90 is required for hyperalgesia triggered via the N-methyl-D-aspartate receptor at the spinal cord level.

=> d history

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FILE 'MEDLINE, CAPLUS, LIFESCI, EMBASE, USPATFULL, BIOSIS' ENTERED AT 17:13:20 ON 25 FEB 2003
L1 2 S HALOETHANE AND PAIN
L2 67441 S HALOTHANE
L3 2608 S L2 AND PAIN
L4 475 S L3 AND INTRATHECAL?
L5 2 S L2 AND PSD93
L6 2 DUP REM L5 (0 DUPLICATES REMOVED)

=> dup rem l4
PROCESSING COMPLETED FOR L4
L7 373 DUP REM L4 (102 DUPLICATES REMOVED)

=> d l7 ibib abs 1-10

L7 ANSWER 1 OF 373 USPATFULL
ACCESSION NUMBER: 2003:51697 USPATFULL
TITLE: 2-(substituted-phenyl)amino-imidazoline derivatives

Clark, Robin Douglas, Palo Alto, CA, UNITED STATES
Jahangir, Alam, San Jose, CA, UNITED STATES
Kowalczyk, Bruce Andrew, Redwood City, CA, UNITED STATES
Lopez-Tapia, Francisco Javier, Fremont, CA, UNITED STATES
Muehldorf, Alexander Victor, Sunnyvale, CA, UNITED STATES
O'Yang, Counde, Sunnyvale, CA, UNITED STATES
Sun, Thomas Weitao, Fremont, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003036655	A1	20030220
APPLICATION INFO.:	US 2002-159589	A1	20020531 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 2000-666065, filed on 19 Sep 2000, ABANDONED Division of Ser. No. US 1998-137507, filed on 20 Aug 1998, GRANTED, Pat. No. US 6184242

	NUMBER	DATE
PRIORITY INFORMATION:	US 1998-89916P	19980619 (60)
	US 1998-88015P	19980604 (60)
	US 1997-57808P	19970904 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ROCHE BIOSCIENCE, 3401 HILLVIEW AVENUE, INTELLECTUAL PROPERTY LAW DEPT., MS A2-250, PALO ALTO, CA, 94304-9819
NUMBER OF CLAIMS: 65
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 3417
AB This invention relates to IP receptor antagonists selected from the group of compounds represented by Formula I: ##STR1##

where:

R.sup.1 is a group represented by formula (A), (B) or (C); ##STR2##

d other substituents as defined in the specification, and their pharmaceutically acceptable ts or crystal forms thereof; and pharmaceutical compositions containing them; and methods their use as therapeutic agents.

L7 ANSWER 2 OF 373 USPATFULL
ACCESSION NUMBER: 2003:51547 USPATFULL
TITLE: Signal transduction pathway component polynucleotides, polypeptides, antibodies and methods based thereon
INVENTOR(S): Barash, Steven C., Rockville, MD, UNITED STATES
Ni, Jian, Germantown, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
Rosen, Craig A., Laytonsville, MD, UNITED STATES
Young, Paul E., Berkeley, CA, UNITED STATES
Rohrschneider, Larry R., Seattle, WA, UNITED STATES
PATENT ASSIGNEE(S): Human Genome Sciences, Inc., Rockville, MD, UNITED STATES, 20850 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003036505	A1	20030220
APPLICATION INFO.:	US 2001-955999	A1	20010920 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-234997P	20000925 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 24363
AB The present invention relates to newly identified human polynucleotides

vectors, host cells, antibodies, and recombinant methods for producing human antigens. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating disorders related to these novel human antigens.

L7 ANSWER 3 OF 373 USPATFULL

ACCESSION NUMBER: 2003:45474 USPATFULL
TITLE: Polynucleotide encoding a novel human potassium channel beta-subunit, K+betaM2
INVENTOR(S): Chang, Han, Princeton Junction, NY, UNITED STATES
Chen, Jian, Princeton, NJ, UNITED STATES
Feder, John, Belle Mead, NJ, UNITED STATES
Jackson, Donald, Lawrenceville, NJ, UNITED STATES
Lee, Liana, North Brunswick, NJ, UNITED STATES
Ramanathan, Chandra S., Wallingford, CT, UNITED STATES
Siemers, Nathan O., Pennington, NJ, UNITED STATES
Carroll, Pamela, Princeton, NJ, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003032786	A1	20030213	
APPLICATION INFO.:	US 2002-56884	A1	20020124	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-263872P	20010124 (60)
	US 2001-269794P	20010214 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: STEPHEN B. DAVIS, BRISTOL-MYERS SQUIBB COMPANY, PATENT DEPARTMENT, P O BOX 4000, PRINCETON, NJ, 08543-4000
NUMBER OF CLAIMS: 25
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 13633

AB The present invention provides novel polynucleotides encoding K+betaM2 polypeptides, fragments and homologues thereof. Also provided are vectors, host cells, antibodies, and recombinant and synthetic methods for producing said polypeptides. The invention further relates to diagnostic and therapeutic methods for applying these novel K+betaM2 polypeptides to the diagnosis, treatment, and/or prevention of various diseases and/or disorders related to these polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of the polynucleotides and polypeptides of the present invention.

L7 ANSWER 4 OF 373 USPATFULL

ACCESSION NUMBER: 2003:45464 USPATFULL
TITLE: Polynucleotide encoding a novel human potassium channel beta-subunit, K+Mbeta1
INVENTOR(S): Feder, John N., Belle Mead, NJ, UNITED STATES
Lee, Liana, North Brunswick, NJ, UNITED STATES
Chen, Jian, Princeton, NJ, UNITED STATES
Jackson, Donald, Lawrenceville, NJ, UNITED STATES
Ramanathan, Chandra, Wallingford, CT, UNITED STATES
Siemers, Nathan, Pennington, NJ, UNITED STATES
Chang, Han, Princeton Junction, NJ, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2003032776	A1	20030213	
APPLICATION INFO.:	US 2001-40805	A1	20011101	(10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-245366P	20001102 (60)
	US 2000-257851P	20001221 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: STEPHEN B. DAVIS, BRISTOL-MYERS SQUIBB COMPANY, PATENT DEPARTMENT, P O BOX 4000, PRINCETON, NJ, 08543-4000
NUMBER OF CLAIMS: 35
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)

AB The present invention provides novel polynucleotides encoding K+Mbeta1 polypeptides, fragments and homologues thereof. Also provided are vectors, host cells, antibodies, and recombinant and synthetic methods for producing said polypeptides. The invention further relates to diagnostic and therapeutic methods for applying these novel K+Mbeta1 polypeptides to the diagnosis, treatment, and/or prevention of various diseases and/or disorders related to these polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of the polynucleotides and polypeptides of the present invention.

L7 ANSWER 5 OF 373 USPATFULL

ACCESSION NUMBER: 2003:38356 USPATFULL

TITLE: 125 human secreted proteins

INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES
Feng, Ping, Gaithersburg, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
Ebner, Reinhard, Gaithersburg, MD, UNITED STATES
Olsen, Henrik S., Gaithersburg, MD, UNITED STATES
Ni, Jian, Germantown, MD, UNITED STATES
Wei, Ying-Fei, Berkeley, CA, UNITED STATES
Soppet, Daniel R., Centreville, VA, UNITED STATES
Moore, Paul A., Germantown, MD, UNITED STATES
Kyaw, Hla, Frederick, MD, UNITED STATES
LaFleur, David W., Washington, DC, UNITED STATES
Shi, Yanggu, Gaithersburg, MD, UNITED STATES
Janat, Fouad, Westerly, RI, UNITED STATES
Endress, Gregory A., Florence, MA, UNITED STATES
Carter, Kenneth C., North Potomac, MD, UNITED STATES
Birse, Charles E., North Potomac, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003028003	A1	20030206
APPLICATION INFO.:	US 2001-974879	A1	20011012 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2001-818683, filed on 28 Mar 2001, PENDING Continuation of Ser. No. US 1999-305736, filed on 5 May 1999, PENDING Continuation-in-part of Ser. No. WO 1998-US23435, filed on 4 Nov 1998, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-239893P	20001013 (60)
	US 1997-64911P	19971107 (60)
	US 1997-64912P	19971107 (60)
	US 1997-64983P	19971107 (60)
	US 1997-64900P	19971107 (60)
	US 1997-64988P	19971107 (60)
	US 1997-64987P	19971107 (60)
	US 1997-64908P	19971107 (60)
	US 1997-64984P	19971107 (60)
	US 1997-64985P	19971107 (60)
	US 1997-66094P	19971117 (60)
	US 1997-66100P	19971117 (60)
	US 1997-66089P	19971117 (60)
	US 1997-66095P	19971117 (60)
	US 1997-66090P	19971117 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

NUMBER OF CLAIMS: 23

EXEMPLARY CLAIM: 1

NUMBER OF DRAWINGS: 3 Drawing Page(s)

LINE COUNT: 36277

AB The present invention relates to novel human secreted proteins and isolated nucleic acids containing the coding regions of the genes encoding such proteins. Also provided are vectors, host cells, antibodies, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating diseases, disorders, and/or conditions related to these novel human secreted proteins.

ACCESSION NUMBER: 2003:38352 USPATFULL
TITLE: 143 human secreted proteins
INVENTOR(S): Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
Moore, Paul A., Germantown, MD, UNITED STATES
Young, Paul E., Gaithersburg, MD, UNITED STATES
Komatsoulis, George A., Silver Spring, MD, UNITED STATES
Birse, Charles E., North Potomac, MD, UNITED STATES
Duan, Roxanne D., Bethesda, MD, UNITED STATES
Florence, Kimberly A., Rockville, MD, UNITED STATES
Soppet, Daniel R., Centreville, VA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003027999	A1	20030206
APPLICATION INFO.:	US 2001-986480	A1	20011108 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 2000-US12788, filed on 11 May 2000, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-134068P	19990513 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
LINE COUNT: 29687
AB The present invention relates to novel human secreted proteins and isolated nucleic acids containing the coding regions of the genes encoding such proteins. Also provided are vectors, host cells, antibodies, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating diseases, disorders, and/or conditions related to these novel human secreted proteins.

L7 ANSWER 7 OF 373 USPATFULL
ACCESSION NUMBER: 2003:38163 USPATFULL
TITLE: Nicotine receptor ligands
INVENTOR(S): Efange, S. Mbua Ngale, Plymouth, MN, UNITED STATES
PATENT ASSIGNEE(S): Regents of the University of Minnesota (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003027810	A1	20030206
APPLICATION INFO.:	US 2001-997718	A1	20011130 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. WO 2000-US15348, filed on 2 Jun 2000, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-137099P	19990602 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SCHWEGMAN, LUNDBERG, WOESSNER & KLUTH, P.A., P.O. BOX 2938, MINNEAPOLIS, MN, 55402
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Page(s)
LINE COUNT: 1953
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides nicotine receptor agonists of formula I:
##STR1##

wherein R.sub.1, x, y, and n have any of the values given in the specification, or a pharmaceutically acceptable salt thereof, as well as pharmaceutical compositions comprising such a compound or salt, methods for preparing such a compound or salt, and methods for modulating (e.g. antagonizing or activating) nicotine receptors with such a compound or salt.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 8 OF 373 USPATFULL
ACCESSION NUMBER: 2003:38129 USPATFULL
TITLE: 29 human cancer associated proteins
INVENTOR(S): Roschke, Viktor, Rockville, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003027776	A1	20030206
APPLICATION INFO.:	US 2001-23896	A1	20011221 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 2000-US23794, filed on 30 Aug 2000, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-152296P	19990903 (60)
	US 1999-158003P	19991006 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
LINE COUNT: 23049
AB This invention relates to newly identified cancer related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "cancer antigens", or alternatively "cancer related proteins", and the use of such cancer antigens for detecting disorders related to the tissues where these cancer antigens are expressed, particularly the presence of cancer and cancer metastases. This invention relates to cancer antigens as well as vectors, host cells, antibodies directed to cancer antigens and the recombinant methods and synthetic methods for producing the same. Also provided are diagnostic methods for detecting, treating, preventing and/or prognosing disorders related to the tissues where these cancer antigens are expressed, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of cancer antigens of the invention. The present invention further relates to inhibiting the production and function of the polypeptides of the present invention.

L7 ANSWER 9 OF 373 USPATFULL
ACCESSION NUMBER: 2003:37652 USPATFULL
TITLE: 19 human secreted proteins
INVENTOR(S): Fiscella, Michele, Bethesda, MD, UNITED STATES
Wei, Ping, Brookeville, MD, UNITED STATES
LaFleur, David W., Washington, DC, UNITED STATES
Olsen, Henrik S., Gaithersburg, MD, UNITED STATES
Baker, Kevin P., Darnestown, MD, UNITED STATES
Ebner, Reinhard, Gaithersburg, MD, UNITED STATES
Komatsoulis, George A., Silver Spring, MD, UNITED STATES
Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
Duan, Roxanne D., Bethesda, MD, UNITED STATES
Young, Paul E., Gaithersburg, MD, UNITED STATES
Florence, Kimberly A., Rockville, MD, UNITED STATES
Moore, Paul A., Germantown, MD, UNITED STATES
Birse, Charles E., North Potomac, MD, UNITED STATES
Ni, Jian, Rockville, MD, UNITED STATES
Soppet, Daniel R., Centreville, VA, UNITED STATES
Shi, Yanggu, Gaithersburg, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003027297	A1	20030206
APPLICATION INFO.:	US 2001-832129	A1	20010411 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 2000-US28664, filed on 17 Oct 2000, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-163085P	19991102 (60)
	US 1999-172411P	19991217 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION

ROCKVILLE, MD, 20850
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
LINE COUNT: 16487
AB The present invention relates to novel human secreted proteins and isolated nucleic acids containing the coding regions of the genes encoding such proteins. Also provided are vectors, host cells, antibodies, and recombinant methods for producing human secreted proteins. The invention further relates to diagnostic and therapeutic methods useful for diagnosing and treating diseases, disorders, and/or conditions related to these novel human secreted proteins.

L7 ANSWER 10 OF 373 USPATFULL
ACCESSION NUMBER: 2003:30977 USPATFULL
TITLE: Method for treating neuropathic ***pain*** and pharmaceutical preparation therefor
INVENTOR(S): Lavand'Homme, Patricia, Brussel, BELGIUM

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003022926	A1	20030130
APPLICATION INFO.:	US 2002-141532	A1	20020507 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-289063P	20010507 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: KNOBBE MARTENS OLSON & BEAR LLP, 2040 MAIN STREET, FOURTEENTH FLOOR, IRVINE, CA, 92614
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Page(s)
LINE COUNT: 827
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a method for a sustained treatment and/or prophylaxis of neuropathic ***pain*** in mammal comprising administering by peripheral nerve injection a neuropathic ***pain*** relieving composition comprising an alpha-2-adrenergic agonist.

The invention further relates to the use of an alpha-2-adrenergic agonist for the preparation of an injectable medicament for the sustained treatment and/or prophylaxis of neuropathic ***pain*** in mammal by peripheral nerve block.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d history

(FILE 'HOME' ENTERED AT 17:12:53 ON 25 FEB 2003)

FILE 'MEDLINE, CAPLUS, LIFESCI, EMBASE, USPATFULL, BIOSIS' ENTERED AT 17:13:20 ON 25 FEB 2003
L1 2 S HALOETHANE AND PAIN
L2 67441 S HALOTHANE
L3 2608 S L2 AND PAIN
L4 475 S L3 AND INTRATHECAL?
L5 2 S L2 AND PSD93
L6 2 DUP REM L5 (0 DUPLICATES REMOVED)
L7 373 DUP REM L4 (102 DUPLICATES REMOVED)

=> s l7 and anesthesia
L8 216 L7 AND ANESTHESIA

=> d l8 ibib abs 200-216

L8 ANSWER 200 OF 216 USPATFULL
ACCESSION NUMBER: 1998:124545 USPATFULL
TITLE: Opioid antagonists and methods of their use
INVENTOR(S): Grandy, David K., Portland, OR, United States
Grisel, Judith E., Portland, OR, United States
Mogil, Jeffrey S., Vancouver, WA, United States
PATENT ASSIGNEE(S): Oregon Health Sciences University, Portland, OR, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5821219		19981013
APPLICATION INFO.:	US 1995-553058		19951103 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1995-514451, filed on 11 Aug 1995
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Walsh, Stephen
ASSISTANT EXAMINER: Basham, Daryl K.
LEGAL REPRESENTATIVE: Klarquist Sparkman Campbell Leigh & Whinston, LLP
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 24 Drawing Figure(s); 14 Drawing Page(s)
LINE COUNT: 2203
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to a novel mammalian anti-opioid receptor protein (OFQR), peptide ligands (such as OFQ) that bind to OFQR, and methods of using the OFQ peptide and analogues to reverse the physiologic effects of opiates such as morphine. The isolation, characterization and pharmacological use of the endogenous peptide ligand is described. A particular embodiment of the OFQ peptide is a heptadecapeptide having an FGGF aminoterminal motif. The peptide specifically binds to an OFQ receptor protein heterologously expressed in mammalian cells. The peptide does not bind with high affinity to .mu., .delta. or .kappa. receptors, but it antagonizes opioid mediated effects (such as analgesia and hypothermia) without increasing nociceptive sensitivity. Tyrosine substitution variants of the peptide ligand specifically bind to the opioid receptor and can be radioiodinated. Also provided are methods of making such peptide ligands and OFQR antagonists, and methods of using the ligands for diagnostic and therapeutic uses and for the identification of other naturally-occurring or synthetic opioid receptor ligands.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 201 OF 216 USPATFULL
ACCESSION NUMBER: 1998:104405 USPATFULL
TITLE: Methods for coextruding immunoisolatory implantable vehicles with a biocompatible jacket and a biocompatible matrix core
INVENTOR(S): Dionne, Keith E., Rehoboth, MA, United States
Emerich, Dwaine F., Providence, RI, United States
Hoffman, Diane, Cambridge, MA, United States
Sanberg, Paul R., Spring Hill, FL, United States
Christenson, Lisa, New Haven, CT, United States
Hegre, Orion D., Green Valley, AZ, United States
Scharp, David W., St. Louis, MO, United States
Lacy, Paul E., Webster Grove, MO, United States
Aebischer, Patrick, Lutry, Switzerland
Vasconcellos, Alfred V., Cranston, RI, United States
Lysaght, Michael J., E. Greenwich, RI, United States
Gentile, Frank T., Warwich, RI, United States
PATENT ASSIGNEE(S): Brown University Research Foundation, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5800829		19980901
APPLICATION INFO.:	US 1995-449274		19950524 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1994-179151, filed on 10 Jan 1994 which is a continuation-in-part of Ser. No. US 1991-693403, filed on 25 Apr 1991, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Bawa, Raj
LEGAL REPRESENTATIVE: Elrifi, Ivor R.Mintz, Levin
NUMBER OF CLAIMS: 27
EXEMPLARY CLAIM: 6
NUMBER OF DRAWINGS: 15 Drawing Figure(s); 9 Drawing Page(s)
LINE COUNT: 3898
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A method of making an immunoisolatory vehicle comprised of a core comprising living cells dispersed in a biocompatible matrix is disclosed, the cells being capable of secreting a biologically active product or of providing a metabolic or immunologic function to an

biocompatible, permselective thermoplastic or hydrogel, said jacket being free of said cells, comprising coextruding a suspension comprising said cells dispersed in a precursor matrix material comprising extracellular matrix components or a biocompatible hydrogel precursor, and a solution of a biocompatible jacket precursor from a nested dual-bore extrusion nozzle, wherein the suspension of (a) is coextruded from the inner bore and the solution of (b) is coextruded from the outer bore of the nozzle, to form said jacket as the solution of (b) and the suspension of (a) arc coextruded; and exposing the vehicle to a treatment that forms a core comprising a volume of at least 1 .mu.l and at least 10.sup.4 cells and comprising a biocompatible matrix from the precursor matrix of solution (a).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 202 OF 216 USPATFULL
ACCESSION NUMBER: 1998:104404 USPATFULL
TITLE: Implantable biocompatible immunoisolatory vehicle for delivery of selected therapeutic products
INVENTOR(S): Dionne, Keith E., Rehoboth, MA, United States
Emerich, Dwaine F., Providence, RI, United States
Hoffman, Diane, Cambridge, MA, United States
Sanberg, Paul R., Spring Hill, FL, United States
Christenson, Lisa, New Haven, CT, United States
Hegre, Orion D., Green Valley, AZ, United States
Scharp, David W., St. Louis, MO, United States
Lacy, Paul E., Webster Grove, MO, United States
Aebischer, Patrick, Lutry, Switzerland
Vasconcellos, Alfred V., Cranston, RI, United States
Lysaght, Michael J., E. Greenwich, RI, United States
Gentile, Frank T., Warwich, RI, United States
PATENT ASSIGNEE(S): Brown University Research Foundation, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5800828		19980901
APPLICATION INFO.:	US 1994-179151		19940110 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1991-692403, filed on 25 Apr 1991, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Bawa, Raj
LEGAL REPRESENTATIVE: Elrifi, Ivor R.Mintz, Levin
NUMBER OF CLAIMS: 43
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Figure(s); 9 Drawing Page(s)
LINE COUNT: 3914
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Immunoisolatory vehicles having a core and a surrounding jacket are disclosed, the core having a volume in excess of 1 .mu.l and at least about 10.sup.4 living cells capable of secreting a biologically active product or of providing a biological function to a patient, the cells dispersed in a biocompatible matrix formed of a hydrogel or an extracellular matrix component, and the external jacket being permselective, biocompatible and having a molecular weight cutoff permitting passage of molecules between the patient and the core through said jacket to provide said biological product or function.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 203 OF 216 USPATFULL
ACCESSION NUMBER: 1998:101409 USPATFULL
TITLE: Implantable biocompatible immunoisolatory vehicle for delivery of selected therapeutic products
INVENTOR(S): Dionne, Keith E., Rehoboth, MA, United States
Emerich, Dwaine F., Providence, RI, United States
Hoffman, Diane, Cambridge, MA, United States
Sanberg, Paul R., Spring Hill, FL, United States
Christenson, Lisa, New Haven, CT, United States
Hegre, Orion D., Green Valley, AZ, United States
Scharp, David W., St. Louis, MO, United States
Lacy, Paul E., Webster Grove, MO, United States
Aebischer, Patrick, Lutry, Switzerland
Vasooncellos, Alfred V., Cranston, RI, United States
Lysaght, Michael J., Greenwich, RI, United States

PATENT ASSIGNEE(S): Brown University Research Foundation, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5798113		19980825
APPLICATION INFO.:	US 1995-449524		19950524 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1994-179151, filed on 10 Jan 1994 which is a continuation-in-part of Ser. No. US 1991-692403, filed on 25 Apr 1991, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Bawa, Raj
LEGAL REPRESENTATIVE: Elrifi, Ivor R., Levin, Mintz
NUMBER OF CLAIMS: 33
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Figure(s); 9 Drawing Page(s)
LINE COUNT: 3901
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A method of providing a biologically active molecule or metabolic or immunologic function to a patient, comprising implanting into the body of the patient at least one immunoisolatory vehicle comprising a core comprising a volume in excess of 1 .mu.l and at least about 10.sup.4 living cells dispersed in a biocompatible matrix formed of a hydrogel or extracellular matrix components, said cells being capable of secreting a biologically active product or of providing a metabolic or immunologic function to the patient; and an external jacket surrounding said core, said jacket being formed from a thermoplastic or hydrogel, said jacket being free of said cells projecting externally therefrom, said jacket being biocompatible and having a molecular weight cutoff permitting passage of molecules between the patient and the core through said jacket to provide said biologically active product of function.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 204 OF 216 USPATFULL
ACCESSION NUMBER: 1998:98888 USPATFULL
TITLE: Stable omega conopetide formulations
INVENTOR(S): Amstutz, Gary Arthur, San Jose, CA, United States
Bowersox, Stephen Scott, Menlo Park, CA, United States
Gohil, Kishorchandra, Richmond, CA, United States
Adriaenssens, Peter Isadore, Mountain View, CA, United States
Kristipati, Ramasharma, Fremont, CA, United States
PATENT ASSIGNEE(S): Neurex Corporation, Menlo Park, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5795864		19980818
APPLICATION INFO.:	US 1995-496847		19950627 (8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Davenport, Avis M.
LEGAL REPRESENTATIVE: Dehlinger, Peter J., Stratford, Carol A.
NUMBER OF CLAIMS: 7
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 18 Drawing Figure(s); 12 Drawing Page(s)
LINE COUNT: 1877
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed are formulations effective to stabilize omega conotoxin peptide preparations at elevated temperatures. Novel omega conopeptides also form part of the invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 205 OF 216 USPATFULL
ACCESSION NUMBER: 1998:69048 USPATFULL
TITLE: Use of kainic acid antagonists to prevent toxic side effects of NMDA antagonists
INVENTOR(S): Olney, John W., 1 Lorenzo La., St. Louis, MO, United States 63124

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5767130		19980616

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1992-877839, filed on 1 May 1992 which is a continuation-in-part of Ser. No. US 1990-467139, filed on 18 Jan 1990, now abandoned which is a continuation-in-part of Ser. No. US 1989-424548, filed on 20 Oct 1989, now patented, Pat. No. US 5034400
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Weddington, Kevin E.
LEGAL REPRESENTATIVE: Kelly, Patrick D.
NUMBER OF CLAIMS: 16
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Figure(s); 1 Drawing Page(s)
LINE COUNT: 1795
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention discloses that kainic acid receptor antagonists (KA antagonists) can act as "safener" agents to reduce or prevent adverse side effects caused by NMDA antagonists. NMDA antagonists can reduce excitotoxic brain damage due to stroke, cardiac arrest, asphyxia, etc., but they also cause toxic damage to certain types of neurons, as well as psychotomimetic effects such as hallucinations. Co-administration of a KA antagonist can (1) reduce or prevent such undesired side effects, and (2) increase the extent of neuronal protection provided to the CNS, beyond the levels of protection that can be provided by NMDA antagonists alone, or non-NMDA antagonists alone. Therefore, co-administration of a KA antagonist allows NMDA antagonists to be used more safely and effectively.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 206 OF 216 USPATFULL
ACCESSION NUMBER: 1998:31136 USPATFULL
TITLE: Inhibitors of adenosine monophosphate deaminase
INVENTOR(S): Erion, Mark D., Del Mar, CA, United States
Bookser, Brett C., Solana Beach, CA, United States
Kasibhatla, Srinivas Rao, San Diego, CA, United States
Gruber, Harry E., Rancho Santa Fe, CA, United States
PATENT ASSIGNEE(S): Gensia Sicor Inc., San Diego, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5731432		19980324
APPLICATION INFO.:	US 1994-192154		19940203 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1993-12841, filed on 3 Feb 1993
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Gupta, Yogendra N.
LEGAL REPRESENTATIVE: Lyon & Lyon LLP
NUMBER OF CLAIMS: 41
EXEMPLARY CLAIM: 1
LINE COUNT: 2952
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel diazepine derivatives which selectively inhibit adenosine monophosphate deaminase and methods of preparing these compounds are provided. These compounds are useful in treating certain conditions in vivo which may be ameliorated by increased local concentrations of adenosine.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 207 OF 216 USPATFULL
ACCESSION NUMBER: 97:96845 USPATFULL
TITLE: Use of adenosine compounds for autonomic nervous system attenuation
INVENTOR(S): Fukunaga, Atsuo F., 5411 Little Bow Rd., Rancho Palos Verdes, CA, United States 90274

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5679649		19971021
APPLICATION INFO.:	US 1995-458981		19950602 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1995-437080, filed on 5 May 1995 which is a continuation of Ser. No. US 1994-203670, filed on 28 Feb 1994, now abandoned which

25 Jun 1993, now abandoned which is a continuation of Ser. No. US 1991-756480, filed on 9 Sep 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-521529, filed on 10 May 1990, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Kight, John
ASSISTANT EXAMINER: Crane, L. Eric
LEGAL REPRESENTATIVE: Fulwider Patton Lee & Utecht, LLP
NUMBER OF CLAIMS: 5
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Figure(s); 9 Drawing Page(s)
LINE COUNT: 1302
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of inducing ***anesthesia*** , sedation, analgesia, hypothermia, and reduced stress by administering an effective amount of an adenosine compound to a mammal. It also provides a method for preserving donor organs in vivo by contacting them with an adenosine compound, as well as a method for preparing organ recipients for transplant.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 208 OF 216 USPATFULL
ACCESSION NUMBER: 97:94223 USPATFULL
TITLE: Therapeutic use of adenosine compounds as surgical anesthetics
INVENTOR(S): Fukunaga, Atsuo F., 5411 Little Bow Rd., Rancho Palos Verdes, CA, United States 90274

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5677290		19971014
APPLICATION INFO.:	US 1995-437080		19950505 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1994-203670, filed on 28 Feb 1994, now abandoned which is a continuation of Ser. No. US 1993-83214, filed on 25 Jun 1993, now abandoned which is a continuation of Ser. No. US 1991-756480, filed on 9 Sep 1991, now abandoned which is a continuation-in-part of Ser. No. US 1990-521529, filed on 10 May 1990, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Kunz, Gary L.
ASSISTANT EXAMINER: Crane, L. Eric
LEGAL REPRESENTATIVE: Fulwider Patton Lee & Utecht, LLP
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1,12,17,20,22
NUMBER OF DRAWINGS: 12 Drawing Figure(s); 9 Drawing Page(s)
LINE COUNT: 1588
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of inducing ***anesthesia*** , sedation, analgesia, hypothermia, and reduced stress by administering an effective amount of an adenosine compound to a mammal. It also provides a method for preserving donor organs in vivo by contacting them with an adenosine compound, as well as a method for preparing organ recipients for transplant.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 209 OF 216 USPATFULL
ACCESSION NUMBER: 97:16066 USPATFULL
TITLE: Use of alpha-2 adrenergic drugs to prevent adverse effects of NMDA receptor hypofunction (NRH)
INVENTOR(S): Olney, John W., Ladue, MO, United States
Farber, Nuri B., University City, MO, United States
PATENT ASSIGNEE(S): Washington University, St. Louis, MO, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5605911		19970225
APPLICATION INFO.:	US 1995-381334		19950131 (8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Nutter, Nathan M.

NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 1935
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods and compositions are disclosed for treating or preventing adverse CNS effects produced by NMDA receptor hypofunction (NRH), including hypofunction induced by NMDA antagonist drugs, and hypofunction occurring as a causative or aggravating factor in schizophrenia. One method of this invention comprises administering an alpha-2 adrenergic (.alpha.2) receptor agonist drug along with an NMDA antagonist drug. The NMDA antagonist drug exerts a primary benefit in reducing excitotoxic brain damage, alleviating neuropathic ***pain*** , or preventing or avoiding tolerance or addiction to various types of drugs. The .alpha.2 agonist drug acts as a secondary or "safener" drug, to prevent the neurotoxic side effects that would be caused by the NMDA antagonist in the absence of the safener drug. Another method disclosed herein involves the use of an .alpha.2 agonist drug, by itself, to combat a different and naturally-occurring form of NMDA receptor hypofunction which occurs as a causative or aggravating mechanism in people suffering from schizophrenia. Although .alpha.2 agonists are usually not effective in treating long-standing cases of chronic schizophrenia, where pathological changes in the brain have already reached or approached maximal levels, .alpha.2 agonists can be administered early in the illness, such as at the first signs of schizophrenic illness, and continuously or intermittently thereafter to prevent the development or worsening of pathological brain changes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 210 OF 216 USPATFULL
ACCESSION NUMBER: 96:118666 USPATFULL
TITLE: Omega conopeptide compositions
INVENTOR(S): Justice, Alan, Sunnyvale, CA, United States
Singh, Tejinder, Palo Alto, CA, United States
Gohil, Kishor C., Richmond, CA, United States
Valentino, Karen L., San Carlos, CA, United States
Miljanich, George P., Redwood City, CA, United States
PATENT ASSIGNEE(S): Neurex Corporation, Menlo Park, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5587454		19961224
APPLICATION INFO.:	US 1993-49794		19930415 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1991-814759, filed on 30 Dec 1991, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Davenport, Avis M.
LEGAL REPRESENTATIVE: Stratford, Carol A., Dehlinger, Peter J.
NUMBER OF CLAIMS: 3
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 51 Drawing Figure(s); 27 Drawing Page(s)
LINE COUNT: 2510
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are novel omega conotoxin peptides effective in producing analgesia.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 211 OF 216 USPATFULL
ACCESSION NUMBER: 96:46072 USPATFULL
TITLE: NMDA-blocking pharmaceuticals
INVENTOR(S): Mechoulam, Raphael, Jerusalem, Israel
Sokolovsky, Mordechai, Tel Aviv, Israel
Kloog, Yoel, Hertzlyia, Israel
Biegon, Anat, Tel Aviv, Israel
PATENT ASSIGNEE(S): Ramot University Authority for Applied Research and Industrial Development Ltd., Tel Aviv, Israel (non-U.S. corporation)
Yissum Research Development Company of the Hebrew University in Jerusalem, Jerusalem, Israel (non-U.S. corporation)
Pharmos Corp., New York, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5521215		19960528
APPLICATION INFO.:	US 1994-192886		19940207 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1992-865088, filed on 8 Apr 1992, now patented, Pat. No. US 5284867 which is a continuation of Ser. No. US 1990-609588, filed on 6 Nov 1990, now abandoned

	NUMBER	DATE
PRIORITY INFORMATION:	IL 1989-92238	19891107

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Chan, Nicky
LEGAL REPRESENTATIVE: Pennie & Edmonds
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 3
NUMBER OF DRAWINGS: 18 Drawing Figure(s); 10 Drawing Page(s)
LINE COUNT: 1572
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Pharmaceutical compositions are described for preventing neurotoxicity, crising as active ingredient the stereospecific (+) enantiomer, having (3S,4S) configuration of .DELTA..sup.6 tetrahydrocannabinol type compounds. The compositions are particularly effective in alleviating and even preventing neurotoxicity due to acute injuries to the central nervous system, including mechanical trauma, compromised or reduced blood supply as may occur in cardiac arrest or stroke, or poisonings. They are also effective in the treatment of certain chronic degenerative diseases characterized by gradual neuronal loss.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 212 OF 216 USPATFULL
ACCESSION NUMBER: 94:99895 USPATFULL
TITLE: Method of producing analgesia
INVENTOR(S): Justice, Alan, Sunnyvale, CA, United States
Singh, Tejinder, Palo Alto, CA, United States
Gohil, Kishor C., Richmond, CA, United States
Valentino, Karen L., San Carlos, CA, United States
PATENT ASSIGNEE(S): Neurex Corporation, Menlo Park, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5364842		19941115
APPLICATION INFO.:	US 1993-81863		19930623 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1991-814759, filed on 30 Dec 1991, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Lee, Lester L.
ASSISTANT EXAMINER: Davenport, A. M.
LEGAL REPRESENTATIVE: Dehlinger, Peter J., Stratford, Carol A.
NUMBER OF CLAIMS: 4
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 34 Drawing Figure(s); 20 Drawing Page(s)
LINE COUNT: 1751
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A method of producing analgesia and enhancing opiate analgesia is disclosed. The method includes administering TVIA (SNX-185) or MVIIA (SNX-111) omega-conopeptide, or derivative thereof which is characterized by its ability to (a) inhibit voltage-gated calcium channels selectively in neuronal tissue, as evidenced by the peptide's ability to inhibit electrically stimulated contraction of the guinea pig ileum, and (b) bind to omega-conopeptide MVIIA binding sites present in neuronal tissue.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 213 OF 216 USPATFULL
ACCESSION NUMBER: 91:90594 USPATFULL
TITLE: Compositions and method for treating painful, inflammatory or allergic disorders
INVENTOR(S): Bernstein, Joel E., Deerfield, IL, United States
PATENT ASSIGNEE(S): Cisco Limited Partnership, Lincolnshire, IL, United

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5063060		19911105
APPLICATION INFO.:	US 1989-452476		19891219 (7)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Page, Thurman K.
ASSISTANT EXAMINER: Hulina, Amy
LEGAL REPRESENTATIVE: Jones, Day, Reavis & Pogue
NUMBER OF CLAIMS: 14
EXEMPLARY CLAIM: 1
LINE COUNT: 242
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to a method of treating painful, inflammatory or allergic disorders comprising treatment with an effective amount of a composition comprising cis-8-methyl-N-vanillyl-6-nonenamide. The invention also relates to compositions for use in the inventive method.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 214 OF 216 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2003:100937 BIOSIS
DOCUMENT NUMBER: PREV200300100937
TITLE: Pre-Emptive Analgesic Effect of General ***Anesthesia*** , Spinal ***Anesthesia*** and Peripheral Nerve Block in Neonatal Rats.
AUTHOR(S): Qiu, Chunyuan (1); Matjasko, Jane (1); Malinow, Andrew M. (1)
CORPORATE SOURCE: (1) Anesthesiology, University of California, Irvine, CA, USA USA
SOURCE: Anesthesiology Abstracts of Scientific Papers Annual Meeting, (2002) No. 2001, pp. Abstract No. A-1288. http://www.asa-abstracts.com. cd-rom.
Meeting Info.: 2001 Annual Meeting of the American Society of Anesthesiologists New Orleans, LA, USA October 13-17, 2001 American Society of Anesthesiologists Inc.
.
DOCUMENT TYPE: Conference
LANGUAGE: English

AB Introduction: Perioperative ***pain*** is commonly seen in pediatric inpatients (1). One promising approach to manage perioperative ***pain*** in children is pre-emptive analgesia. However, Little is known about the pre-emptive analgesic effect of different anesthetics in neonatal human and rat. Complete Freund's adjuvant(CFA) can induce inflammation and persistent ***pain*** in both adult rats and rat pups(2,3), Characterized by hyperalgesia, allodynia and central sensitization. The animal model has been widely used for studying ***pain*** behavior and responses to ***pain*** therapy in neonatal rat. We used this animal model to test the hypothesis and efficiency of pre-emptive analgesia by different anesthetics in rat pups. We also used Fos positive neuron in the spinal cord as a marker of central sensitization.(4,5) Methods: 18-22 postnatal day rat were used. Normal saline(NS)(10ul) or CFA(10ul,1:1 oil/saline) was injected into one hind paw. Paw withdrawal latency(PWL)by thermal stimulation was measured in all animals except regional ***anesthesia*** groups before and 2 hours after CFA or NS hind paw injection. General ***anesthesia*** (2% ***halothane***); ***intrathecal*** bupivacaine (50ug in 10ul) or femoral and sciatic nerve block (total 500 ug in 0.2ml) were applied before CFA injection. After 2 hours of CFA stimulation, the rats were perfused with 4% paraformaldahyde and L4-5 was processed for Fos protein staining. Fos immunoreactivity was determined and compared between different ***anesthesia*** groups. Results: CFA injection resulted in behavioral hyperalgesia within 2 hours of stimulation as determined by PWL from 11.0+-1.7s to 5.4+-1.1s. Spinal Fos expression increased from 4.7+-0.4 to 23.6+-1.5. General ***anesthesia*** delayed CFA induced PWL to 7.2+-0.8s and suppressed spinal superficial Fos expression by 39.8%(14.4+-2.1) Peripheral nerve block abolished CFA induced FOS expression whereas ***intrathecal*** local anesthetic partially blocked the Fos expression in the superficial dorsal horn. Conclusion: ***Pain*** response in neonatal rat is well developed. Aggressive pediatric ***pain*** management is strongly suggested. Pre-emptive analgesia and its effectiveness depended on ***anesthesia*** techniques. Partial ***pain*** relieve was observed after a period of exposure to inhalation agent probably due to decreased central sensitization. Complete blocking of Fos expression by peripheral nerve

analgesia. Effect of ***intrathecal*** local anesthetics as a pre-emptive analgesia need further study because ***intrathecal*** local anesthetics only partially block the expression of Fos in the spinal cord.

L8 ANSWER 215 OF 216 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:89830 BIOSIS
DOCUMENT NUMBER: PREV200100089830
TITLE: The effect of ***intrathecal*** dexmedetomidine on induction of Fos-like immunoactivity in the spinal dorsal horn in a rat postoperative ***pain*** model.
AUTHOR(S): Shimode, N. (1); Tanimoto, M.; Tashiro, T.; Fukuoka, T.; Kondo, E.; Noguchi, K.
CORPORATE SOURCE: (1) Hyogo College of Medicine, Hyogo Japan
SOURCE: Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-354.2. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000 Society for Neuroscience
. ISSN: 0190-5295.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Clonidine, an alpha2-adrenergic agonist produces antinociception when injected epidurally or ***intrathecally*** . Dexmedetomidine, more specific alpha2-adrenoceptor agonists than clonidine, decreases MAC(minimum alveolar concentration) of ***halothane*** with dose-dependent manner. Fos is an immediate early gene product induced in spinal dorsal horn by noxious stimuli. A surgical incision in planter aspect of the rat hindpaw has been used as a postoperative ***pain*** model. In this study, we examined Fos induction in the spinal dorsal horn in this model, and investigated the effect of ***intrathecal*** dexmedetomidine on this Fos expression. Under ***halothane*** (2%) ***anesthesia*** , Sprague-Dawley male rats (300-350g) were injected saline or dexmedetomidine (0.1, 0.3, 1, 3, or 10 mug) ***intrathecally*** . Thirty minutes later, they received surgical incision described above and were sutured with 5-0 nylon. Two hours after the noxious surgical procedure, all rats were intracardially perfused with 4% paraformaldehyde. The L5 segment of spinal cord was dissected out and cut 30 mum transverse sections using a cryostat. These sections were immunohistochemically stained for Fos protein. In saline group, Fos positive neurons were observed mainly in the superficial laminae (I, II) and, to a lesser extent, in laminae III-V. The number of Fos-like immunoreactive neurons decreased in laminae I-V by dexmedetomidine pretreatment with dose-dependent manner. We conclude that ***intrathecal*** dexmedetomidine suppress neural response of spinal neurons to noxious stimuli in respect to Fos expression.

L8 ANSWER 216 OF 216 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1996:322537 BIOSIS
DOCUMENT NUMBER: PREV199699044893
TITLE: Antagonism of the antinocifensive action of ***halothane*** by ***intrathecal*** administration of GABA-A receptor antagonists.
AUTHOR(S): Mason, Peggy; Owens, Casey A.; Hammond, Donna L.
CORPORATE SOURCE: Dep. Pharmacol. and Physiol. Sci., Committee on Neurobiol., Univ. Chicago, MC 0926, 947 East 58th St., Chicago, IL 60637 USA
SOURCE: Anesthesiology (Hagerstown), (1996) Vol. 84, No. 5, pp. 1205-1214.
ISSN: 0003-3022.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Background: The hind brain and the spinal cord, regions that contain high concentrations of gamma-aminobutyric acid (GABA) and GABA receptors, have been implicated as sites of action of inhalational anesthetics. Previous studies have established that general anesthetics potentiate the effects of gamma-aminobutyric acid at the GABA-A receptor. It was therefore hypothesized that the suppression of nocifensive movements during ***anesthesia*** is due to an enhancement of GABA-A receptor-mediated transmission within the spinal cord. Methods: Rats in which an ***intrathecal*** catheter had been implanted 1 week earlier were anesthetized with ***halothane*** . Core temperature was maintained at a steady level. After MAC determination, the concentration of ***halothane*** was adjusted to that at which the rats last moved in response to tail clamping. Saline, a GABA-A, a GABA-B, or a glycine

to move in response to application of the tail clamp was redetermined 5 min later, after which the ***halothane*** concentration was increased by 0.2%. Response latencies to application of the noxious stimulus were measured at 7-min intervals during the subsequent 35 min. To determine whether these antagonists altered baseline response latencies by themselves, another experiment was conducted in which the concentration of ***halothane*** was not increased after ***intrathecal*** administration of GABA-A receptor antagonists. Results: ***Intrathecal*** administration of the GABA-A receptor antagonists bicuculline (0.3 mu-g) or picrotoxin (0.3, 1.0 mu-g) antagonized the suppression of nocifensive movement produced by the small increase in ***halothane*** concentration. in contrast, the antinocifensive effect of the increase in ***halothane*** concentration was not attenuated by the GABA-B receptor antagonist CGP 35348 or the glycine receptor antagonist strychnine. By themselves, the GABA-A receptor antagonists did not alter response latency in rats anesthetized with sub-MAC concentrations of ***halothane*** . Conclusions: ***Intrathecal*** administration of bicuculline or picrotoxin, at doses that do not change the latency to pinch-evoked movement when administered alone, antagonized the suppression of noxious-evoked movement produced by ***halothane*** concentrations equal to or greater than MAC. These results suggest that enhancement of GABA-A receptor-mediated transmission within the spinal cord contributes to ***halothane*** 's ability to suppress nocifensive movements.

=> d ibib kwic 211

L8 ANSWER 211 OF 216 USPATFULL
ACCESSION NUMBER: 96:46072 USPATFULL
TITLE: NMDA-blocking pharmaceuticals
INVENTOR(S): Mechoulam, Raphael, Jerusalem, Israel
Sokolovsky, Mordechai, Tel Aviv, Israel
Kloog, Yoel, Hertzlyia, Israel
Biegon, Anat, Tel Aviv, Israel
PATENT ASSIGNEE(S): Ramot University Authority for Applied Research and Industrial Development Ltd., Tel Aviv, Israel (non-U.S. corporation)
Yissum Research Development Company of the Hebrew University in Jerusalem, Jerusalem, Israel (non-U.S. corporation)
Pharmos Corp., New York, NY, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5521215		19960528
APPLICATION INFO.:	US 1994-192886		19940207 (8)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1992-865088, filed on 8 Apr 1992, now patented, Pat. No. US 5284867 which is a continuation of Ser. No. US 1990-609588, filed on 6 Nov 1990, now abandoned

	NUMBER	DATE
PRIORITY INFORMATION:	IL 1989-92238	19891107

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Chan, Nicky
LEGAL REPRESENTATIVE: Pennie & Edmonds
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 3
NUMBER OF DRAWINGS: 18 Drawing Figure(s); 10 Drawing Page(s)
LINE COUNT: 1572
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
DETD . . . for rectal administration. Liquid forms may be prepared for oral administration or for injection, the term including subcutaneous, transdermal, intravenous, ***intrathecal*** , and other parenteral routes of administration. The liquid compositions include aqueous solutions, with or without organic cosolvents, aqueous or oil. . .
DETD . . . of the compositions of the present invention. The term administration as used herein encompasses oral, parenteral, intravenous, intramuscular, subcutaneous, transdermal, ***intrathecal*** , rectal and intranasal administration.
DETD Animals (Sprague-Dawley rats weighing 300-400 g) were fasted overnight but were allowed free access to water. ***Anesthesia*** was induced

and was maintained with 2% ***halothane*** and 70% nitrous oxide during the surgical procedures. Atropine sulfate (0.04 mg, i.p.) was injected. The right femoral artery and. . .

DETD Steady state monitor, drug administration and MCAo. Following these surgical procedures, the inspired ***halothane*** was discontinued to avoid the effect of ***halothane*** on systemic blood pressure and CBF. ***Anesthesia*** was maintained with 70% nitrous oxide and 30% oxygen. Thirty minutes after discontinuation of ***halothane*** , measurement of the preischemic physiological variables, CBF, MAP, and pulse rate was begun. Steady-state baseline values were recorded before the. . .

DETD . . . scores in stroke model in gerbils (normal score, 0)

Neurological behavior

	Score(s)
Normal	0
Sleepy/lethargic	1
Hyperactive	2
Circling/Ptosis	3
Jumping	4
Tossing seizures/Ophistolonus	5
Tonic convulsion	6
Coma, weak ***pain*** response	7
Coma, no ***pain*** response	8
Death	9

Modification of Rudolphi's Clinical scoring method.

DETD . . . supplied by Anilab (Hulda, Israel) were used in this study. They were anesthetized using Pentothal (Abbott, Italy) for induction, with ***Halothane*** (ICI Pharmaceuticals, England), in a mixture of 70% N.sub.2 and 30% 0.sub.2 for maintenance.

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=> d history

     (FILE 'HOME' ENTERED AT 17:12:53 ON 25 FEB 2003)

     FILE 'MEDLINE, CAPLUS, LIFESCI, EMBASE, USPATFULL, BIOSIS' ENTERED AT
     17:13:20 ON 25 FEB 2003
L1             2 S HALOETHANE AND PAIN
L2         67441 S HALOTHANE
L3          2608 S L2 AND PAIN
L4           475 S L3 AND INTRATHECAL?
L5             2 S L2 AND PSD93
L6             2 DUP REM L5 (0 DUPLICATES REMOVED)
L7           373 DUP REM L4 (102 DUPLICATES REMOVED)
L8           216 S L7 AND ANESTHESIA

=> s haloethane (p) intrathecal?
L9             0 HALOETHANE (P) INTRATHECAL?

=> s psd93 and nmda
L10           13 PSD93 AND NMDA

=> dup rem l10
PROCESSING COMPLETED FOR L10
L11            12 DUP REM L10 (1 DUPLICATE REMOVED)

=> d l11

L11  ANSWER 1 OF 12  USPATFULL
AN     2002:85548  USPATFULL
TI     Inhibition of interaction of ***PSD93*** and PSD95 with nNOS and
         ***NMDA*** receptors
IN     Tao, Yuanxiang, Baltimore, MD, UNITED STATES
       Johns, Roger A., Reistertown, MD, UNITED STATES
PI     US 2002045590      A1   20020418
AI     US 2001-853895     A1   20010514 (9)
PRAI   US 2000-242580P     20001023 (60)
DT     Utility
FS     APPLICATION
LN.CNT 1513
```

NCL *NCLM: 514/044.000
IC [7]
ICM: A61K048-00
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d l11 2-12

L11 ANSWER 2 OF 12 CAPLUS COPYRIGHT 2003 ACS
AN 2002:535636 CAPLUS
DN 137:346662
TI Nucleus-specific expression of ***NMDA*** receptor-associated postsynaptic density proteins in primate thalamus
AU Clinton, Sarah M.; Meador-Woodruff, James H.
CS Department of Psychiatry and Mental Health Research Institute, University of Michigan Medical School, Ann Arbor, MI, 48109-0720, USA
SO Thalamus & Related Systems (2002), 1(4), 303-316
CODEN: TRSHBY; ISSN: 1472-9288
PB Elsevier Science Ltd.
DT Journal
LA English
RE.CNT 70 THERE ARE 70 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 3 OF 12 CAPLUS COPYRIGHT 2003 ACS
AN 2001:850924 CAPLUS
DN 135:366767
TI Inhibition of interaction of ***psd93*** and psd95 with neuronal nitric oxide synthase and ***NMDA*** receptors
IN Johns, Roger A.; Tao, Yuanxiang
PA The Johns Hopkins University, USA
SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001087285	A2	20011122	WO 2001-US15372	20010514
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	US 2002045590	A1	20020418	US 2001-853895	20010514
PRAI	US 2000-203894P	P	20000512		
	US 2000-242580P	P	20001023		

L11 ANSWER 4 OF 12 CAPLUS COPYRIGHT 2003 ACS
AN 2001:318984 CAPLUS
DN 135:58927
TI PSD-93 knock-out mice reveal that neuronal MAGUKs are not required for development or function of parallel fiber synapses in cerebellum
AU McGee, Aaron W.; Topinka, J. Rick; Hashimoto, Kouichi; Petralia, Ronald S.; Kakizawa, Sho; Kauer, Frederick; Aguilera-Moreno, Andrea; Wenthold, Robert J.; Kano, Masanobu; Bredt, David S.
CS Department of Physiology and Programs in Biomedical Sciences and Neuroscience, University of California at San Francisco School of Medicine, San Francisco, CA, 94143-0444, USA
SO Journal of Neuroscience (2001), 21(9), 3085-3091
CODEN: JNRSDS; ISSN: 0270-6474
PB Society for Neuroscience
DT Journal
LA English
RE.CNT 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 5 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 2001:547003 BIOSIS
DN PREV200100547003
TI Altered expression of ***NMDA*** receptor-related post-synaptic density proteins in thalamus of schizophrenia.

Meador-Woodruff, J. H. (1)
CS (1) Mental Health Research Institute, University Michigan, Ann Arbor, MI USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 1193. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience San Diego, California, USA November 10-15, 2001
ISSN: 0190-5295.
DT Conference
LA English
SL English

L11 ANSWER 6 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 2001:487322 BIOSIS
DN PREV200100487322
TI Expression and developmental changes of PSD-95 and PSD-93 in rat spinal cord.
AU Tao, Y. X. (1); Levine, C. F. (1); Fang, M. (1); Gonzalez, J. A. (1); Tao, F. (1); Huganir, R. L.; Bredt, D. S.; Johns, R. A. (1)
CS (1) Dept Anesthesiology, Johns Hopkins Univ Sch Med, Baltimore, MD USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 416. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience San Diego, California, USA November 10-15, 2001
ISSN: 0190-5295.
DT Conference
LA English
SL English

L11 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2003 ACS
AN 2001:342623 CAPLUS
DN 135:74417
TI Electron microscopic immunocytochemical detection of PSD-95, PSD-93, SAP-102, and SAP-97 at postsynaptic, presynaptic, and nonsynaptic sites of adult and neonatal rat visual cortex
AU Aoki, Chiye; Miko, Ilona; Oviedo, Hysell; Mikeladze-Dvali, Tamara; Alexandre, Lucien; Sweeney, Neal; Bredt, David S.
CS Center for Neural Science, New York University, New York, NY, 10003, USA
SO Synapse (New York, NY, United States) (2001), 40(4), 239-257
CODEN: SYNAET; ISSN: 0887-4476
PB Wiley-Liss, Inc.
DT Journal
LA English
RE.CNT 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 8 OF 12 USPATFULL
AN 2000:106055 USPATFULL
TI CAPON: a protein associated with neuronal nitric oxide synthase
IN Snyder, Solomon H., Baltimore, MD, United States
Jaffrey, Samie R., Baltimore, MD, United States
PA The Johns Hopkins University, Baltimore, MD, United States (U.S. corporation)
PI US 6103872 20000815
AI US 1998-10998 19980122 (9)
DT Utility
FS Granted
LN.CNT 1968
INCL INCLM: 530/350.000
INCLS: 530/326.000; 530/327.000; 530/328.000
NCL NCLM: 530/350.000
NCLS: 530/326.000; 530/327.000; 530/328.000
IC [7]
ICM: C07K007-06
ICS: C07K007-08; C07K014-47
EXF 530/300; 530/324; 530/325; 530/326; 530/327; 530/328; 530/350
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 9 OF 12 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 1
AN 2000:115989 CAPLUS
DN 132:234751
TI A developmental change in ***NMDA*** receptor-associated proteins at hippocampal synapses
AU Sans, Nathalie; Petralia, Ronald S.; Wang, Ya-Xian; Blahos, Jaroslav; Hell, Johannes W.; Wenthold, Robert J.
CS Laboratory of Neurochemistry, National Institute on Deafness and Other Communication Disorders, National Institutes of Health, Bethesda,

SO Journal of Neuroscience (2000), 20(3), 1260-1271
CODEN: JNRSDS; ISSN: 0270-6474
PB Society for Neuroscience
DT Journal
LA English
RE.CNT 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 10 OF 12 CAPLUS COPYRIGHT 2003 ACS
AN 1999:167564 CAPLUS
DN 131:85876
TI Distinct spatiotemporal expression of mRNAs for the PSD-95/SAP90 protein family in the mouse brain
AU Fukaya, Masahiro; Ueda, Hiroshi; Yamauchi, Kohei; Inoue, Yoshiro; Watanabe, Masahiko
CS Department of Anatomy, School of Medicine, Hokkaido University, Sapporo, 060-8638, Japan
SO Neuroscience Research (Shannon, Ireland) (1999), 33(2), 111-118
CODEN: NERADN; ISSN: 0168-0102
PB Elsevier Science Ireland Ltd.
DT Journal
LA English
RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2003 ACS
AN 1998:78074 CAPLUS
DN 128:254216
TI CAPON: a protein associated with neuronal nitric oxide synthase that regulates its interactions with PSD95
AU Jaffrey, Samie R.; Snowman, Adele M.; Eliasson, Mikael J. L.; Cohen, Noam A.; Snyder, Solomon H.
CS School of Medicine, Departments of Neuroscience, Pharmacology and Molecular Sciences, and Psychiatry, The Johns Hopkins University, Baltimore, MD, 21205, USA
SO Neuron (1998), 20(1), 115-124
CODEN: NERNET; ISSN: 0896-6273
PB Cell Press
DT Journal
LA English

L11 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2003 ACS
AN 1996:716663 CAPLUS
DN 126:2233
TI Cloning and characterization of postsynaptic density 93, a nitric oxide synthase interacting protein
AU Brenman, Jay E.; Christopherson, Karen S.; Craven, Sarah E.; McGee, Aaron W.; Bredt, David S.
CS Dep. Physiol., Univ. California at San Francisco Sch. Med., San Francisco, CA, 94143-0444, USA
SO Journal of Neuroscience (1996), 16(23), 7407-7415
CODEN: JNRSDS; ISSN: 0270-6474
PB Society for Neuroscience
DT Journal
LA English

=> d l11 ibib abs tot

L11 ANSWER 1 OF 12 USPATFULL

ACCESSION NUMBER:	2002:85548 USPATFULL		
TITLE:	Inhibition of interaction of ***PSD93*** and PSD95 with nNOS and ***NMDA*** receptors		
INVENTOR(S):	Tao, Yuanxiang, Baltimore, MD, UNITED STATES Johns, Roger A., Reistertown, MD, UNITED STATES		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002045590	A1	20020418
APPLICATION INFO.:	US 2001-853895	A1	20010514 (9)
	NUMBER		DATE
PRIORITY INFORMATION:	US 2000-242580P		20001023 (60)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	APPLICATION		

WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 65
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 1513
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB PSD-95/SAP90 antisense-treated animals not only experience a significant decrease in MAC for isoflurane, but also experience an attenuation in the ***NMDA*** -induced increase in isoflurane MAC. PSD-95/SAP90 appears to mediate the role of the ***NMDA*** receptor in determining the MAC of inhalational anesthetics. Suppression of the expression of PSD-95/SAP90 in the spinal cord significantly attenuates responses to painful stimuli mediated through the N-methyl-D-aspartate receptor activation. In spinal cord neurons PSD-95/SAP90 interacts with the N-methyl-D-aspartate receptor subunits 2A/2B. Activation of the N-methyl-D-aspartate receptor in spinal hyperalgesia results in association of the N-methyl-D-aspartate receptor with PSD-95/SAP90. PSD-95/SAP90 is required for hyperalgesia triggered via the N-methyl-D-aspartate receptor at the spinal cord level.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 2 OF 12 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:535636 CAPLUS
DOCUMENT NUMBER: 137:346662
TITLE: Nucleus-specific expression of ***NMDA*** receptor-associated postsynaptic density proteins in primate thalamus
AUTHOR(S): Clinton, Sarah M.; Meador-Woodruff, James H.
CORPORATE SOURCE: Department of Psychiatry and Mental Health Research Institute, University of Michigan Medical School, Ann Arbor, MI, 48109-0720, USA
SOURCE: Thalamus & Related Systems (2002), 1(4), 303-316
CODEN: TRSHBY; ISSN: 1472-9288
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Thalamic afferents and efferents primarily use the neurotransmitter glutamate, which acts through a variety of ionotropic (***NMDA*** , AMPA, kainate) and metabotropic receptors. The NMDAR is composed of multiple subunits, NR1 and NR2A-D. The obligatory NR1 subunit is expressed as one of eight isoforms, due to the alternative splicing of exons 5, 21, and 22. Each NR1 splice variant is functionally distinct. For instance, alternative splicing of exons 21 and 22 renders two C-terminal variants, which differentially assoc. with NR2 subunits and intracellular mols. such the PSD-95 family of proteins. These PSD proteins play a pivotal role in NMDAR function by linking NMDARs to the cytoskeleton and downstream signal-transducing enzymes that can directly modulate NMDAR function and/or promote NMDAR-assocd. intracellular events. Previous work reported that NR1 is by far the most abundant NMDAR subunit expressed in the primate thalamus. In the current study, the authors extend these findings first by detg. which NR1 isoforms are predominantly expressed in the thalamus. Secondly, the authors characterize the expression of the NMDAR-assocd. PSD mols., such as PSD-95, in the thalamus. Using in situ hybridization, the authors examd. expression of the transcripts encoding NR1 isoforms contg. exons 5, 21, or 22, and transcripts encoding a set of the most well-characterized NMDAR-assocd. PSD proteins (NF-L, ***PSD93*** , PSD95, SAP102, and Yotiao). NR1 exon 22-contg. isoforms are the most abundant subunit transcripts, accounting for 40-50% of the NR1 isoforms expressed in most thalamic nuclei. The authors also found that NF-L is by far the most abundant PSD protein expressed in the thalamus, followed by PSD-95, which is moderately and heterogeneously expressed. SAP102 and PSD-93 were expressed at moderate to low levels, with negligible amts. of Yotiao transcript expression. The PSD-95 family of mols. are crit. for NMDAR function in the cell, and this study is the first to provide a detailed description of the expression of these mols. in primate thalamus. The authors' results demonstrate that NR1 splice variants and assocd. PSD proteins are heterogeneously expressed across the thalamus, which is likely related to the intracellular events that occur in different thalamic nuclei.
REFERENCE COUNT: 70 THERE ARE 70 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 3 OF 12 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:850924 CAPLUS
DOCUMENT NUMBER: 135:366767

with neuronal nitric oxide synthase and ***NMDA*** receptors
INVENTOR(S): Johns, Roger A.; Tao, Yuanxiang
PATENT ASSIGNEE(S): The Johns Hopkins University, USA
SOURCE: PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001087285	A2	20011122	WO 2001-US15372	20010514
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2002045590	A1	20020418	US 2001-853895	20010514
PRIORITY APPLN. INFO.:			US 2000-203894P	P 20000512
			US 2000-242580P	P 20001023

AB PSD-95/SAP90 antisense-treated animals not only experience a significant decrease in min. alveolar concn. (MAC) for isoflurane, but also experience an attenuation in the ***NMDA*** -induced increase in isoflurane MAC. PSD-95/SAP90 appears to mediate the role of the ***NMDA*** receptor in detg. the MAC of inhalational anesthetics. Suppression of the expression of PSD-95/SAP90 in the spinal cord significantly attenuates responses to painful stimuli mediated through the N-methyl-D-aspartate receptor activation. In spinal cord neurons PSD-95/SAP90 interacts with the N-methyl-D-aspartate receptor subunits 2A/2B. Activation of the N-methyl-D-aspartate receptor in spinal hyperalgesia results in assocn. of the N-methyl-D-aspartate receptor with PSD-95/SAP90. PSD-95/SAP90 is required for hyperalgesia triggered via the N-methyl-D-aspartate receptor at the spinal cord level.

L11 ANSWER 4 OF 12 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:318984 CAPLUS
DOCUMENT NUMBER: 135:58927
TITLE: PSD-93 knock-out mice reveal that neuronal MAGUKs are not required for development or function of parallel fiber synapses in cerebellum
AUTHOR(S): McGee, Aaron W.; Topinka, J. Rick; Hashimoto, Kouichi; Petralia, Ronald S.; Kakizawa, Sho; Kauer, Frederick; Aguilera-Moreno, Andrea; Wenthold, Robert J.; Kano, Masanobu; Bredt, David S.
CORPORATE SOURCE: Department of Physiology and Programs in Biomedical Sciences and Neuroscience, University of California at San Francisco School of Medicine, San Francisco, CA, 94143-0444, USA
SOURCE: Journal of Neuroscience (2001), 21(9), 3085-3091
CODEN: JNRSDS; ISSN: 0270-6474
PUBLISHER: Society for Neuroscience
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Membrane-assocd. guanylate kinases (MAGUKs) are abundant postsynaptic d. (PSD)-95/disks large/zona occludens-1 (PDZ)-contg. proteins that can assemble receptors and assocd. signaling enzymes at sites of cell-cell contact, including synapses. PSD-93, a postsynaptic neuronal MAGUK, has three PDZ domains that can bind to specific ion channels, including ***NMDA*** .delta.2 type glutamate receptors, as well as Shaker and inward rectifier type K+ channels, and can mediate clustering of these channels in heterologous cells. Genetic analyses of Drosophila show that MAGUKs play crit. roles in synaptic development because mutations of disks large disrupt the sub-synaptic reticulum and block postsynaptic clustering of Shaker K+ channels. It is uncertain whether MAGUKs play an essential role in the development of central synapses. There are four neuronal MAGUKs with overlapping expression patterns in the mammalian brain; however, we find PSD-93 is the only MAGUK expressed in cerebellar Purkinje neurons. Therefore, we targeted disruption of PSD-93 in mouse. Despite the absence of MAGUK immunoreactivity in Purkinje neurons from the knock-outs, these mice have no structural or functional abnormality in

localization of PSD-93 interacting proteins remain intact at light and electron microscopic levels in the knock-outs. Postsynaptic Purkinje cell responses, monosynaptic climbing fiber innervation, and cerebellar-dependent behaviors are also normal. Our data demonstrate that MAGUK proteins of the PSD-93/95 family are not essential for development of certain central synapses but may instead participate in specialized aspects of synaptic signaling and plasticity.
REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 5 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:547003 BIOSIS
DOCUMENT NUMBER: PREV200100547003
TITLE: Altered expression of ***NMDA*** receptor-related post-synaptic density proteins in thalamus of schizophrenia.
AUTHOR(S): Clinton, S. M. (1); Haroutunian, V. (1); Davis, K. L. (1); Meador-Woodruff, J. H. (1)
CORPORATE SOURCE: (1) Mental Health Research Institute, University Michigan, Ann Arbor, MI USA
SOURCE: Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 1193. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience San Diego, California, USA November 10-15, 2001
ISSN: 0190-5295.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Converging evidence suggests that NMDAR receptor (NMDAR) dysfunction plays a role in the pathophysiology of schizophrenia. Normal NMDAR activation and related intracellular signaling relies on the presence of neighboring receptors, co-factors, and post-synaptic density (PSD)-related proteins. These PSD proteins target NMDARs to the synaptic membrane and facilitate interactions with various intracellular components, and altering this receptor-PSD protein interaction may alter normal NMDAR function. Recently, we reported that NR1 and NR2C NMDAR subunits are abnormally expressed in limbic thalamic nuclei in schizophrenia. Since NMDAR subunit expression is altered in schizophrenic thalamus, we hypothesized that NMDAR-related PSD proteins may also be abnormally expressed. Using in situ hybridization, we examined mRNA expression of NMDAR-related PSD proteins NF-L, ***PSD93*** , PSD95, SAP102, and Yotiao. We detected a apprx30% increase of NF-L and SAP102 expression in schizophrenic thalamus, compared to control ($p<0.01$), but did not detect changes in expression of ***PSD93*** , PSD95, or Yotiao. We are currently using Western Blot analysis to measure the protein levels of NMDAR subunits and related PSD proteins, to test whether protein expression parallels the observed changes in mRNA expression. Altered PSD protein expression may reflect a compensatory change that stems from a primary dysfunction of the NMDAR. Further, these data suggest that glutamatergic dysfunction in schizophrenia may occur at the level of intracellular signaling in addition to receptor expression.

L11 ANSWER 6 OF 12 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:487322 BIOSIS
DOCUMENT NUMBER: PREV200100487322
TITLE: Expression and developmental changes of PSD-95 and PSD-93 in rat spinal cord.
AUTHOR(S): Tao, Y. X. (1); Levine, C. F. (1); Fang, M. (1); Gonzalez, J. A. (1); Tao, F. (1); Huganir, R. L.; Bredt, D. S.; Johns, R. A. (1)
CORPORATE SOURCE: (1) Dept Anesthesiology, Johns Hopkins Univ Sch Med, Baltimore, MD USA
SOURCE: Society for Neuroscience Abstracts, (2001) Vol. 27, No. 1, pp. 416. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience San Diego, California, USA November 10-15, 2001
ISSN: 0190-5295.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
AB We have demonstrated that PSD-95 is critical for ***NMDA*** receptor-mediated spinal hyperalgesia. To further provide morphological support, the present work examined the expression and developmental changes of PSD-95 and its family member, PSD-93, in the spinal cord.

SAP97 were enriched in the spinal cord and other brain regions. PSD-95 and its family members were not detected in the dorsal root ganglia. Immunocytochemistry revealed that PSD-95 was distributed mainly in lamina I of the spinal cord, while PSD-93 was concentrated in both laminae I and II. During postnatal development in the spinal cord, these two proteins exhibited distinct changes in expression. PSD-95 was strongly expressed before postnatal day 10 and showed a substantial decrease by 6 months. However, PSD-93 expression was at a low level prior to postnatal day 5, reached a peak at postnatal day 20 and was slightly reduced by 6 months. Immunoprecipitation experiments demonstrated that both PSD-95 and PSD-93 in the spinal cord interacted with ***NMDA*** receptors. The area-specific expression and distribution of PSD-95 and PSD-93 suggest that PSD-95 and PSD-93 are important in mechanisms of spinal nociceptive processing. Moreover, distinct distribution and developmental changes in PSD-95/SAP90 and PSD-93 expression indicate that they might have specific functions that are critical to synaptic development and signal transduction in the spinal cord.

L11 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:342623 CAPLUS
DOCUMENT NUMBER: 135:74417
TITLE: Electron microscopic immunocytochemical detection of PSD-95, PSD-93, SAP-102, and SAP-97 at postsynaptic, presynaptic, and nonsynaptic sites of adult and neonatal rat visual cortex
AUTHOR(S): Aoki, Chiye; Miko, Ilona; Oviedo, Hysell; Mikeladze-Dvali, Tamara; Alexandre, Lucien; Sweeney, Neal; Bredt, David S.
CORPORATE SOURCE: Center for Neural Science, New York University, New York, NY, 10003, USA
SOURCE: Synapse (New York, NY, United States) (2001), 40(4), 239-257
CODEN: SYNAET; ISSN: 0887-4476
PUBLISHER: Wiley-Liss, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Membrane-assocd. guanylate kinases (MAGUKs) assemble protein complexes at sites of cell-cell contact. At excitatory synapses in brain, MAGUKs localize to the postsynaptic d. (PSD) and interact with N-methyl-D-aspartate (***NMDA***) glutamate receptors and downstream signaling proteins. However, ***NMDA*** receptors are not restricted to the PSDs, as electron microscopic immunocytochem. (EM-ICC) results indicate that ***NMDA*** receptors also occur at nonsynaptic portions of dendrites, perhaps functioning as reserves for rapid insertion into synaptic membranes in response to appropriate synaptic activity. ***NMDA*** receptors also occur in axons, at least in part to support glutamate-dependent enhancement of transmitter release. In this study, a systematic EM-ICC survey was performed to det. whether the distributions of four neuronal MAGUKs-PSD-95, PSD-93, SAP-102, and SAP-97-resemble that of ***NMDA*** receptors. Quant. anal. revealed that the d. of PSD-95 over thick PSDs of asym. axo-spinous synaptic junctions is 2-3-fold the level in the immediately adjacent cytoplasm of spines and terminals, while sym. synapses show no assocn. with PSD-95. Similarly, all four MAGUKs occur over PSDs of spines. However, we also detected MAGUK immunoreactivity, albeit more diffusely, along presynaptic membranes and in the cytoplasm of axons and dendritic shafts. In fact, the overall distribution of PSD-95 within the neuropil is equally prevalent along plasma membranes (including synaptic portions) as in the cytoplasm, away from plasma membranes. These results suggest that MAGUKs have dual roles: to maintain receptors at synapses and to regulate shuttling of receptors between nonsynaptic and synaptic sites.
REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 8 OF 12 USPATFULL
ACCESSION NUMBER: 2000:106055 USPATFULL
TITLE: CAPON: a protein associated with neuronal nitric oxide synthase
INVENTOR(S): Snyder, Solomon H., Baltimore, MD, United States
Jaffrey, Samie R., Baltimore, MD, United States
PATENT ASSIGNEE(S): The Johns Hopkins University, Baltimore, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6103872		20000815

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Schwartzman, Robert A.
LEGAL REPRESENTATIVE: Banner & Witcoff, Ltd.
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 17 Drawing Figure(s); 9 Drawing Page(s)
LINE COUNT: 1968
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Nitric oxide (NO) produced by neuronal nitric oxide synthase (nNOS) is important for N-methyl-D-aspartate (***NMDA***) receptor-dependent neurotransmitter release, neurotoxicity, and cyclic-GMP elevations. The coupling of ***NMDA*** receptor-mediated calcium influx and nNOS activation is postulated to be due to a physical coupling of the receptor and the enzyme by an intermediary adaptor protein PSD95, through a unique PDZ-PDZ domain interaction between PSD95 and nNOS. Here we report the identification of a novel nNOS associated protein, CAPON, which is highly enriched in brain and has numerous colocalizations with nNOS. CAPON interacts with the NNOS PDZ domain through its C-terminus. CAPON competes with PSD95 for interaction with nNOS, and overexpression of CAPON results in a loss of PSD95/nNOS complexes in transfected cells. CAPON influences nNOS by regulating its ability to associate with PSD95/ ***NMDA*** receptor complexes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 9 OF 12 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 1
ACCESSION NUMBER: 2000:115989 CAPLUS
DOCUMENT NUMBER: 132:234751
TITLE: A developmental change in ***NMDA*** receptor-associated proteins at hippocampal synapses
AUTHOR(S): Sans, Nathalie; Petralia, Ronald S.; Wang, Ya-Xian; Blahos, Jaroslav; Hell, Johannes W.; Wenthold, Robert J.
CORPORATE SOURCE: Laboratory of Neurochemistry, National Institute on Deafness and Other Communication Disorders, National Institutes of Health, Bethesda, Bethesda, MD, 20892, USA
SOURCE: Journal of Neuroscience (2000), 20(3), 1260-1271
CODEN: JNRSDS; ISSN: 0270-6474
PUBLISHER: Society for Neuroscience
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The membrane-assocd. guanylate kinases [Chapsyn-110/postsynaptic d.-93 (PSD-93), synapse-assocd. protein-90 (SAP-90)/PSD-95, and SAP-102] are believed to cluster and anchor ***NMDA*** receptors at the synapse and to play a role in signal transduction. The authors have investigated the developmental changes in expression of these proteins in rat hippocampus using biochem. analyses and quant. immunogold electron microscopy. At postnatal day 2 (P2), SAP-102 was highly expressed, whereas PSD-93 and PSD-95 were low. SAP-102 expression increased during the first week, stayed stable through P35, and showed a reduced expression at 6 mo. From P2 through 6 mo, PSD-93 and PSD-95 increased. For PSD-95, the percent of labeled synapses increased almost threefold with age, whereas the no. of gold particles per labeled synapse did not change significantly, suggesting that the increase in PSD-95 is attributable primarily to an increase in the no. of synapses contg. PSD-95. In contrast, for SAP-102, both percent labeled synapses and the no. of gold particles per labeled synapse decreased during this time. From Western blots of hippocampus and immunogold anal. of CA1 synapses, the high expression of NR2B at P2 coincides with the high level of SAP-102 at synapses, whereas the later expression of NR2A coincides with that of PSD-93 and PSD-95. To det. whether the changes in PSD-93/95 and SAP-102 reflect preferred assocns. with NR2A and NR2B, resp., the authors measured co-immunopptn. in the adult hippocampus. These studies suggest that there is a preference for complexes of NR2A/PSD-93/95 and NR2B/SAP-102. These results indicate that individual receptor-assocd. proteins may have specific functions that are crit. to synapse development.
REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 10 OF 12 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1999:167564 CAPLUS
DOCUMENT NUMBER: 131:85876
TITLE: Distinct spatiotemporal expression of mRNAs for the PSD-95/SAP90 protein family in the mouse brain

Inoue, Yoshiro; Watanabe, Masahiko
CORPORATE SOURCE: Department of Anatomy, School of Medicine, Hokkaido University, Sapporo, 060-8638, Japan
SOURCE: Neuroscience Research (Shannon, Ireland) (1999), 33(2), 111-118
CODEN: NERADN; ISSN: 0168-0102
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB PSD-95 (SAP90), SAP102, and Chapsyn-110 (PSD-93) are members of the membrane-assocd. guanylate kinase family, and interact with N-methyl-D-aspartate (***NMDA***) receptor NR2A (GluR.epsilon.1) and NR2B (GluR.epsilon.2) subunits and with Shaker-type K+channel subunits to cluster into a channel complex. Here, the authors examd. their expression in developing and adult mouse brains by in situ hybridization with antisense oligonucleotide probes. PSD-95 and SAP102 mRNAs were prominently expressed at embryonic day 13 (E13) in the mantle zone of various brain regions, where ***NMDA*** receptor NR2B subunit mRNA was expressed at high levels. In the early postnatal period when active synaptogenesis takes place, both mRNAs became elevated and concd. in the telencephalon and cerebellar granular layer, where NR2A and/or NR2B subunit mRNAs were abundantly expressed. Chapsyn-110 mRNA was, although at low levels, found over the mantle zone of embryonic brains, and the level was progressively increased in the telencephalon starting at perinatal stages. The spatial and temporal correlations in the brain in vivo suggested that the PSD-95/SAP90 protein family can interact with ***NMDA*** receptor subunits to cluster them into a channel complex at both synaptic and nonsynaptic sites before, during, and after synaptogenic stages.
REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L11 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1998:78074 CAPLUS
DOCUMENT NUMBER: 128:254216
TITLE: CAPON: a protein associated with neuronal nitric oxide synthase that regulates its interactions with PSD95
AUTHOR(S): Jaffrey, Samie R.; Snowman, Adele M.; Eliasson, Mikael J. L.; Cohen, Noam A.; Snyder, Solomon H.
CORPORATE SOURCE: School of Medicine, Departments of Neuroscience, Pharmacology and Molecular Sciences, and Psychiatry, The Johns Hopkins University, Baltimore, MD, 21205, USA
SOURCE: Neuron (1998), 20(1), 115-124
CODEN: NERNET; ISSN: 0896-6273
PUBLISHER: Cell Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Nitric oxide (NO) produced by neuronal nitric oxide synthase (nNOS) is important for N-methyl-D-aspartate (***NMDA***) receptor-dependent neurotransmitter release, neurotoxicity, and cGMP elevations. The coupling of ***NMDA*** receptor-mediated calcium influx and nNOS activation is postulated to be due to a phys. coupling of the receptor and the enzyme by an intermediary adaptor protein, PSD95, through a unique PDZ-PDZ domain interaction between PSD95 and nNOS. Here, the authors report the identification of a novel nNOS-assocd. protein, CAPON, which is highly enriched in brain and has numerous colocalizations with nNOS. CAPON interacts with the nNOS PDZ domain through its C terminus. CAPON competes with PSD95 for interaction with nNOS, and overexpression of CAPON results in a loss of PSD95/nNOS complexes in transfected cells. CAPON may influence nNOS by regulating its ability to assoc. with PSD95/ ***NMDA*** receptor complexes.

L11 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 1996:716663 CAPLUS
DOCUMENT NUMBER: 126:2233
TITLE: Cloning and characterization of postsynaptic density 93, a nitric oxide synthase interacting protein
AUTHOR(S): Brenman, Jay E.; Christopherson, Karen S.; Craven, Sarah E.; McGee, Aaron W.; Bredt, David S.
CORPORATE SOURCE: Dep. Physiol., Univ. California at San Francisco Sch. Med., San Francisco, CA, 94143-0444, USA
SOURCE: Journal of Neuroscience (1996), 16(23), 7407-7415
CODEN: JNRSDS; ISSN: 0270-6474
PUBLISHER: Society for Neuroscience
DOCUMENT TYPE: Journal

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•AB   Nitric oxide (NO) formation in brain is regulated by the
      calcium/calmodulin dependence of neuronal NO synthase (nNOS).  Calcium
      influx through   ***NMDA***  -type glutamate receptors is efficiently
      coupled to nNOS activity, whereas many other intracellular calcium
      pathways are poorly coupled.  To elucidate possible mechanisms responsible
      for this coupling, we performed yeast two-hybrid screening to identify
      proteins that interact with nNOS.  Two nNOS interacting proteins were
      identified: the postsynaptic d. proteins PSD-93 and PSD-95.  Here, we
      report the cloning and characterization of PSD-93.  PSD-93 is expressed in
      discrete neuronal populations as well as in specific non-neuronal cells,
      and it exhibits complex mol. diversity attributable to tissue-specific
      alternative splicing.  PSD-93, like PSD-95, binds to nNOS and to the
        ***NMDA***   receptor 2B.  PSD-93, however, is unique among PSD-95/SAP-90
      family members in its expression in Purkinje neuron cell bodies and
      dendrites.  We also demonstrate that the PDZ domain at the N terminus of
      nNOS is required, but it is not sufficient for interaction with PSD-93/95.
      Given that PSD-93 and PSD-95 each contain multiple potential binding sites
      for nNOS and the   ***NMDA***   receptor, complexes involving oligomers of
      PSD-93/95 may help account for the functional as well as the phys.
      coupling of nNOS to   ***NMDA***   receptors.


=> d history

     (FILE 'HOME' ENTERED AT 17:12:53 ON 25 FEB 2003)

     FILE 'MEDLINE, CAPLUS, LIFESCI, EMBASE, USPATFULL, BIOSIS' ENTERED AT
     17:13:20 ON 25 FEB 2003
L1             2 S HALOETHANE AND PAIN
L2         67441 S HALOTHANE
L3          2608 S L2 AND PAIN
L4           475 S L3 AND INTRATHECAL?
L5             2 S L2 AND PSD93
L6             2 DUP REM L5 (0 DUPLICATES REMOVED)
L7           373 DUP REM L4 (102 DUPLICATES REMOVED)
L8           216 S L7 AND ANESTHESIA
L9             0 S HALOETHANE (P) INTRATHECAL?
L10           13 S PSD93 AND NMDA
L11           12 DUP REM L10 (1 DUPLICATE REMOVED)

=> s ((psd()93) or (chapsyn()110)) and (pain or anesthe?)
L12            5 ((PSD(W) 93) OR (CHAPSYN(W) 110)) AND (PAIN OR ANESTHE?)

=> dup rem l12
PROCESSING COMPLETED FOR L12
L13            5 DUP REM L12 (0 DUPLICATES REMOVED)

=> d l13

L13  ANSWER 1 OF 5  USPATFULL
AN     2003:30332  USPATFULL
TI     Novel genes encoding proteins having prognostic, diagnostic, preventive,
       therapeutic, and other uses
IN     Fraser, Christopher C., Lexington, MA, UNITED STATES
       Barnes, Thomas M., Brookline, MA, UNITED STATES
       Sharp, John D., Arlington, MA, UNITED STATES
       Kirst, Susan J., Brookline, MA, UNITED STATES
       Myers, Paul S., Cambridge, MA, UNITED STATES
       Leiby, Kevin R., Natick, MA, UNITED STATES
       Holtzman, Douglas A., Jamaica Plain, MA, UNITED STATES
       McCarthy, Sean A., San Diego, CA, UNITED STATES
       Wrighton, Nicholas, Winchester, MA, UNITED STATES
       MacKay, Charles R., Vaucluse, AUSTRALIA
       Goodearl, Andrew D.J., Natick, MA, UNITED STATES
PI     US 2003022279      A1   20030130
AI     US 2001-759130     A1   20010112 (9)
RLI    Continuation-in-part of Ser. No. US 2000-479249, filed on 7 Jan 2000,
       ABANDONED Continuation-in-part of Ser. No. US 2000-559497, filed on 27
       Apr 2000, ABANDONED Continuation-in-part of Ser. No. US 2000-578063,
       filed on 24 May 2000, PENDING Continuation-in-part of Ser. No. US
       1999-333159, filed on 14 Jun 1999, PENDING Continuation-in-part of Ser.
       No. US 2000-596194, filed on 16 Jun 2000, PENDING Continuation-in-part
       of Ser. No. US 1999-342364, filed on 29 Jun 1999, ABANDONED
       Continuation-in-part of Ser. No. US 2000-608452, filed on 30 Jun 2000,
       PENDING Continuation-in-part of Ser. No. US 1999-393996, filed on 10 Sep
       1999, ABANDONED Continuation-in-part of Ser. No. US 2000-602871, filed
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1999-420707, filed on 19 Oct 1999, ABANDONED
DT Utility
FS APPLICATION
LN.CNT 12618
INCL INCLM: 435/069.100
INCLS: 435/320.100; 435/325.000; 514/044.000; 530/350.000; 536/023.200; 800/008.000
NCL NCLM: 435/069.100
NCLS: 435/320.100; 435/325.000; 514/044.000; 530/350.000; 536/023.200; 800/008.000
IC [7]
ICM: A01K067-00
ICS: C07H021-04; C12P021-02; C12N005-06; C07K014-435
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d l13 ibib abs tot

L13 ANSWER 1 OF 5 USPATFULL
ACCESSION NUMBER: 2003:30332 USPATFULL
TITLE: Novel genes encoding proteins having prognostic, diagnostic, preventive, therapeutic, and other uses
INVENTOR(S): Fraser, Christopher C., Lexington, MA, UNITED STATES
Barnes, Thomas M., Brookline, MA, UNITED STATES
Sharp, John D., Arlington, MA, UNITED STATES
Kirst, Susan J., Brookline, MA, UNITED STATES
Myers, Paul S., Cambridge, MA, UNITED STATES
Leiby, Kevin R., Natick, MA, UNITED STATES
Holtzman, Douglas A., Jamaica Plain, MA, UNITED STATES
McCarthy, Sean A., San Diego, CA, UNITED STATES
Wrighton, Nicholas, Winchester, MA, UNITED STATES
MacKay, Charles R., Vaucluse, AUSTRALIA
Goodearl, Andrew D.J., Natick, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003022279	A1	20030130
APPLICATION INFO.:	US 2001-759130	A1	20010112 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-479249, filed on 7 Jan 2000, ABANDONED Continuation-in-part of Ser. No. US 2000-559497, filed on 27 Apr 2000, ABANDONED Continuation-in-part of Ser. No. US 2000-578063, filed on 24 May 2000, PENDING Continuation-in-part of Ser. No. US 1999-333159, filed on 14 Jun 1999, PENDING Continuation-in-part of Ser. No. US 2000-596194, filed on 16 Jun 2000, PENDING Continuation-in-part of Ser. No. US 1999-342364, filed on 29 Jun 1999, ABANDONED Continuation-in-part of Ser. No. US 2000-608452, filed on 30 Jun 2000, PENDING Continuation-in-part of Ser. No. US 1999-393996, filed on 10 Sep 1999, ABANDONED Continuation-in-part of Ser. No. US 2000-602871, filed on 23 Jun 2000, ABANDONED Continuation-in-part of Ser. No. US 1999-420707, filed on 19 Oct 1999, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Jean M. Silveri, Millenium Pharmaceuticals, Inc., 75 Sidney Street, Cambridge, MA, 02139
NUMBER OF CLAIMS: 85
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 361 Drawing Page(s)
LINE COUNT: 12618
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides isolated nucleic acids encoding a variety of proteins having diagnostic, preventive, therapeutic, and other uses. These nucleic and proteins are useful for diagnosis, prevention, and therapy of a number of human and other animal disorders. The invention also provides antisense nucleic acid molecules, expression vectors containing the nucleic acid molecules of the invention, host cells into which the expression vectors have been introduced, and non-human transgenic animals in which a nucleic acid molecule of the invention has been introduced or disrupted. The invention still further provides isolated polypeptides, fusion polypeptides, antigenic peptides and antibodies. Diagnostic, screening, and therapeutic methods using compositions of the invention are also provided. The nucleic acids and polypeptides of the present invention are useful as modulating agents in regulating a variety of cellular processes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L13 ANSWER 2 OF 5 USPATFULL

ACCESSION NUMBER: 2002:322511 USPATFULL
TITLE: Novel genes encoding proteins having diagnostic, preventive, therapeutic and other uses
INVENTOR(S): McCarthy, Sean A., San Diego, CA, UNITED STATES
Fraser, Christopher C., Lexington, MA, UNITED STATES
Sharp, John D., Arlington, MA, UNITED STATES
Barnes, Thomas M., Brookline, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., Cambridge, MA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002182675	A1	20021205
APPLICATION INFO.:	US 2001-42431	A1	20011025 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-578063, filed on 24 May 2000, PENDING Continuation-in-part of Ser. No. US 1999-333159, filed on 14 Jun 1999, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: AKIN, GUMP, STRAUSS, HAUER & FELD, L.L.P., ONE COMMERCE SQUARE, 2005 MARKET STREET, SUITE 2200, PHILADELPHIA, PA, 19103
NUMBER OF CLAIMS: 51
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 95 Drawing Page(s)
LINE COUNT: 9736
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides isolated nucleic acids encoding a variety of proteins having diagnostic, preventive, therapeutic, and other uses. These nucleic and proteins are useful for diagnosis, prevention, and therapy of a number of human and other animal disorders. The invention also provides antisense nucleic acid molecules, expression vectors containing the nucleic acid molecules of the invention, host cells into which the expression vectors have been introduced, and non-human transgenic animals in which a nucleic acid molecule of the invention has been introduced or disrupted. The invention still further provides isolated polypeptides, fusion polypeptides, antigenic peptides and antibodies. Diagnostic, screening, and therapeutic methods utilizing compositions of the invention are also provided. The nucleic acids and polypeptides of the present invention are useful as modulating agents in regulating a variety of cellular processes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L13 ANSWER 3 OF 5 USPATFULL

ACCESSION NUMBER: 2002:266423 USPATFULL
TITLE: Peptides that modulate the interaction of B class ephrins and PDZ domains
INVENTOR(S): Lin, Danny, Scarborough, CANADA
Pawson, Anthony, Toronto, CANADA
Gish, Gerald, East York, CANADA

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002147306	A1	20021010
APPLICATION INFO.:	US 2001-862179	A1	20010521 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	WO 1999-CA1101	19991119
	US 1998-109158P	19981120 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ROPES & GRAY, ONE INTERNATIONAL PLACE, BOSTON, MA, 02110-2624
NUMBER OF CLAIMS: 35
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 17 Drawing Page(s)
LINE COUNT: 2332
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to complexes comprising a B class ephrin and a PDZ domain containing protein; peptides that interfere with the interaction of a B class ephrin with a PDZ domain binding site, and a PDZ domain

modulating the interaction of a B class ephrin and a PDZ domain containing protein, and methods for evaluating compounds for their ability to modulate the interaction are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L13 ANSWER 4 OF 5 USPATFULL

ACCESSION NUMBER:	2002:85548 USPATFULL			
TITLE:	Inhibition of interaction of PSD93 and PSD95 with nNOS and NMDA receptors			
INVENTOR(S):	Tao, Yuanxiang, Baltimore, MD, UNITED STATES Johns, Roger A., Reistertown, MD, UNITED STATES			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002045590	A1	20020418	
APPLICATION INFO.:	US 2001-853895	A1	20010514	(9)
	NUMBER		DATE	
PRIORITY INFORMATION:	US 2000-242580P		20001023	(60)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	APPLICATION			
LEGAL REPRESENTATIVE:	BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001			
NUMBER OF CLAIMS:	65			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	4 Drawing Page(s)			
LINE COUNT:	1513			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB PSD-95/SAP90 antisense-treated animals not only experience a significant decrease in MAC for isoflurane, but also experience an attenuation in the NMDA-induced increase in isoflurane MAC. PSD-95/SAP90 appears to mediate the role of the NMDA receptor in determining the MAC of inhalational ***anesthetics*** . Suppression of the expression of PSD-95/SAP90 in the spinal cord significantly attenuates responses to painful stimuli mediated through the N-methyl-D-aspartate receptor activation. In spinal cord neurons PSD-95/SAP90 interacts with the N-methyl-D-aspartate receptor subunits 2A/2B. Activation of the N-methyl-D-aspartate receptor in spinal hyperalgesia results in association of the N-methyl-D-aspartate receptor with PSD-95/SAP90. PSD-95/SAP90 is required for hyperalgesia triggered via the N-methyl-D-aspartate receptor at the spinal cord level.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L13 ANSWER 5 OF 5 USPATFULL

ACCESSION NUMBER:	2002:346979 USPATFULL			
TITLE:	Composition for the detection of signaling pathway gene expression			
INVENTOR(S):	Au-Young, Janice, Berkeley, CA, United States Seilhamer, Jeffrey J., Los Altos Hills, CA, United States			
PATENT ASSIGNEE(S):	Incyte Genomics, Inc., Palo Alto, CA, United States (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6500938	B1	20021231	
APPLICATION INFO.:	US 1998-16434		19980130	(9)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	GRANTED			
PRIMARY EXAMINER:	Marschel, Ardin H.			
LEGAL REPRESENTATIVE:	Incyte Genomics, Inc.			
NUMBER OF CLAIMS:	5			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	0 Drawing Figure(s); 0 Drawing Page(s)			
LINE COUNT:	6180			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to a composition comprising a plurality of polynucleotide probes. The composition can be used as array elements in a microarray. The present invention also relates to a method for selecting polynucleotide probes of the composition.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d kwic 2

L13 ANSWER 2 OF 5 USPATFULL
DETD . . . junction protein ZO-1, vertebrate erythrocyte membrane protein p55, C. elegans protein lin-2, rat protein CASK, and mammalian synaptic proteins SAP90/PSD-95, ***CHAPSYN*** - ***110*** / ***PSD*** - ***93*** , SAP97/DLG1, and SAP102), proteins which interact with vertebrate receptor protein tyrosine kinases (e.g., mammalian cytoplasmic protein Nck and oncoprotein Crk),. . .
DETD [0209] CNS-related disorders include disorders associated with developmental, cognitive, and autonomic neural and neurological processes, such as ***pain*** , appetite, long term memory, and short term memory.
DETD . . . barrier (e.g., CNS infections such as meningitis and encephalitis, aseptic meningitis, metastasis of non-CNS tumor cells into the CNS, various ***pain*** disorders such as migraine, blindness and other vision problems, and CNS-related adverse drug reactions such as head ***pain*** , sleepiness, and confusion). TANGO 273 proteins, nucleic acids encoding them, and agents that modulate activity or expression of either of. . .

=> d ibib 2

L13 ANSWER 2 OF 5 USPATFULL
ACCESSION NUMBER: 2002:322511 USPATFULL
TITLE: Novel genes encoding proteins having diagnostic, preventive, therapeutic and other uses
INVENTOR(S): McCarthy, Sean A., San Diego, CA, UNITED STATES
Fraser, Christopher C., Lexington, MA, UNITED STATES
Sharp, John D., Arlington, MA, UNITED STATES
Barnes, Thomas M., Brookline, MA, UNITED STATES
PATENT ASSIGNEE(S): Millennium Pharmaceuticals, Inc., Cambridge, MA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002182675	A1	20021205
APPLICATION INFO.:	US 2001-42431	A1	20011025 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2000-578063, filed on 24 May 2000, PENDING Continuation-in-part of Ser. No. US 1999-333159, filed on 14 Jun 1999, PENDING
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: AKIN, GUMP, STRAUSS, HAUER & FELD, L.L.P., ONE COMMERCE SQUARE, 2005 MARKET STREET, SUITE 2200, PHILADELPHIA, PA, 19103
NUMBER OF CLAIMS: 51
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 95 Drawing Page(s)
LINE COUNT: 9736
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> e johns r?/au

E1	5		JOHNS R T/AU
E2	25		JOHNS R W/AU
E3	0	-->	JOHNS R?/AU
E4	2		JOHNS RACHEL/AU
E5	1		JOHNS RALPH HOWARD/AU
E6	3		JOHNS RAY/AU
E7	1		JOHNS RAYMOND/AU
E8	2		JOHNS REBECCA/AU
E9	1		JOHNS REBECCA A/AU
E10	1		JOHNS REBECCA L/AU
E11	2		JOHNS REED L/AU
E12	20		JOHNS REGINALD B/AU

=> e johns roger?/au

E1	164		JOHNS ROGER A/AU
E2	3		JOHNS ROGER P/AU
E3	0	-->	JOHNS ROGER?/AU
E4	6		JOHNS ROLF M/AU
E5	1		JOHNS RON H/AU
E6	1		JOHNS RONALD/AU
E7	2		JOHNS RONALD E/AU

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E9          2     JOHNS ROY W/AU
E10         1     JOHNS RUEDIGER/AU
E11         1     JOHNS RUSSELL/AU
E12         1     JOHNS RUSSELL C/AU

=> s e1
L14         164 "JOHNS ROGER A"/AU

=> dup rem l14
PROCESSING COMPLETED FOR L14
L15         116 DUP REM L14 (48 DUPLICATES REMOVED)

=> s l15 and nmda
L16         11 L15 AND NMDA

=> d l16 ibib abs tot

L16  ANSWER 1 OF 11  CAPLUS  COPYRIGHT 2003 ACS
ACCESSION NUMBER:         2001:850924  CAPLUS
DOCUMENT NUMBER:          135:366767
TITLE:                    Inhibition of interaction of psd93 and psd95 with
                          neuronal nitric oxide synthase and   ***NMDA***
                          receptors
INVENTOR(S):                ***Johns, Roger A.***  ; Tao, Yuanxiang
PATENT ASSIGNEE(S):       The Johns Hopkins University, USA
SOURCE:                   PCT Int. Appl., 45 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

      PATENT NO.        KIND  DATE          APPLICATION NO.  DATE
      ---------------   ----  --------      ---------------  --------
      WO 2001087285      A2   20011122      WO 2001-US15372  20010514
          W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
              CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
              GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
              LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT,
              RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US,
              UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
          RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
              DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
              BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
      US 2002045590      A1   20020418      US 2001-853895   20010514
PRIORITY APPLN. INFO.:                     US 2000-203894P  P  20000512
                                           US 2000-242580P  P  20001023

AB    PSD-95/SAP90 antisense-treated animals not only experience a significant
      decrease in min. alveolar concn. (MAC) for isoflurane, but also experience
      an attenuation in the   ***NMDA***  -induced increase in isoflurane MAC.
      PSD-95/SAP90 appears to mediate the role of the   ***NMDA***   receptor in
      detg. the MAC of inhalational anesthetics.  Suppression of the expression
      of PSD-95/SAP90 in the spinal cord significantly attenuates responses to
      painful stimuli mediated through the N-methyl-D-aspartate receptor
      activation.  In spinal cord neurons PSD-95/SAP90 interacts with the
      N-methyl-D-aspartate receptor subunits 2A/2B.  Activation of the
      N-methyl-D-aspartate receptor in spinal hyperalgesia results in assocn. of
      the N-methyl-D-aspartate receptor with PSD-95/SAP90.  PSD-95/SAP90 is
      required for hyperalgesia triggered via the N-methyl-D-aspartate receptor
      at the spinal cord level.

L16  ANSWER 2 OF 11  CAPLUS  COPYRIGHT 2003 ACS
ACCESSION NUMBER:         2001:839105  CAPLUS
DOCUMENT NUMBER:          136:353639
TITLE:                    Knockdown of PSD-95/SAP90 delays the development of
                          neuropathic pain in rats
AUTHOR(S):                Tao, Feng; Tao, Yuan-Xiang; Gonzalez, Julio A.; Fang,
                          Ming; Mao, Peizhong;   ***Johns, Roger A.***
CORPORATE SOURCE:         Department of Anesthesiology and Critical Care
                          Medicine, Johns Hopkins University School of Medicine,
                          Baltimore, MD, 21287-4965, USA
SOURCE:                   NeuroReport (2001), 12(15), 3251-3255
                          CODEN: NERPEZ; ISSN: 0959-4965
PUBLISHER:                Lippincott Williams & Wilkins
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
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receptor-mediated thermal hyperalgesia. To address the role of PSD-95/SAP90 in chronic pain, the present study investigated the effect of the deficiency of PSD-95/SAP90 on nerve injury-induced neuropathic pain. Following unilateral L5 spinal nerve injury, mech. and thermal hyperalgesia developed within 3 days and persisted for 9 days or longer on the injured side. The intrathecal administration of antisense oligodeoxynucleotide specifically against PSD-95/SAP90, but not sense or missense oligodeoxynucleotide, dose-dependently delayed the onset of tactile allodynia and thermal hyperalgesia. These results suggest that PSD-95/SAP90 might be involved in the central mechanisms of the development of chronic neuropathic pain.

REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 3 OF 11 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2001:475912 CAPLUS
DOCUMENT NUMBER: 136:210425
TITLE: Effect of the deficiency of spinal PSD-95/SAP90 on the minimum alveolar anesthetic concentration of isoflurane in rats
AUTHOR(S): Tao, Yuan-Xiang; ***Johns, Roger A.***
CORPORATE SOURCE: Department of Anesthesiology and Critical Care Medicine, Johns Hopkins University School of Medicine, Baltimore, MD, 21287-4965, USA
SOURCE: Anesthesiology (2001), 94(6), 1010-1015
CODEN: ANESAV; ISSN: 0003-3022
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Spinal N-methyl-D-aspartate (***NMDA***) receptor activation was demonstrated to play an important role in the processing of spinal nociceptive information and in the detn. of the min. alveolar anesthetic concn. (MAC) of inhalational anesthetics. Postsynaptic d.-95 (PSD-95)/synapse-assocd. protein-90 (SAP90), a mol. scaffolding protein that binds and clusters the ***NMDA*** receptor preferentially at synapses, was implicated in ***NMDA*** -induced thermal hyperalgesia. The current study investigated the possible involvement of PSD-95/SAP90 in detg. MAC for isoflurane anesthesia. Sprague-Dawley rats were pretreated intrathecally with PSD-95/SAP90 antisense oligodeoxyribonucleotide (ODN), sense ODN, missense ODN, or saline every 24 h for 4 days. After initial baseline detn. of the MAC, ***NMDA*** or saline was injected intrathecally. Ten minutes later, MAC measurement was repeated. The rats also were evaluated for the presence of locomotor dysfunction by intrathecal administration of ***NMDA*** or saline in the saline- and ODN-treated rats. In the groups treated with antisense ODNs, but not in those treated with sense or missense ODNs, there was a significant decrease in isoflurane MAC that was not accompanied by marked changes in either blood pressure or heart rate. In the saline-treated group, intrathecal ***NMDA*** caused an increase in isoflurane MAC. In contrast, in the antisense ODN-treated group, intrathecal ***NMDA*** did not produce a significant change in isoflurane MAC. An ***NMDA*** -induced increase in blood pressure but not heart rate was found in both saline- and antisense ODN-treated groups. Locomotor activity was not changed in any of the treated animals. The results indicate not only a significant decrease in MAC for isoflurane but also an attenuation in the ***NMDA*** -induced increase in isoflurane MAC in the PSD-95/SAP90 antisense-treated animals, which suggests that PSD-95/SAP90 may mediate the role of the ***NMDA*** receptor in detg. the MAC of inhalational anesthetics.

REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2001:434853 CAPLUS
DOCUMENT NUMBER: 135:29155
TITLE: Cyclic GMP-dependent protein kinase isoform-specific inhibition for treatment of pain and reduction of anesthetic threshold
INVENTOR(S): ***Johns, Roger A.*** ; Tao, Yuanxiang
PATENT ASSIGNEE(S): The Johns Hopkins University, USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001041752	A2	20010614	WO 2000-US33195	20001208
WO 2001041752	A3	20020912		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 2001031750	A1	20011018	US 2000-731876	20001208
US 6476007	B2	20021105		
US 2003022866	A1	20030130	US 2002-183635	20020628

PRIORITY APPLN. INFO.: US 1999-170260P A1 19991208
US 2000-731876 A3 20001208

AB Several lines of evidence have shown a role for the nitric oxide (NO)/cyclic guanosine monophosphate (cGMP) signaling pathway in the development of spinal hyperalgesia. However, the roles of effectors for cGMP are not fully understood in the processing of pain in the spinal cord. CGMP-dependent protein kinase (PKG) I.alpha. but not PKGI.beta. was localized in the neuronal bodies and processes, and was distributed primarily in the superficial laminae of the spinal cord. Intrathecal administration of an inhibitor of PKGI.alpha., Rp-8-[(4-Chlorophenyl)thio]-cGMPS triethylamine, produces significant antinociception. Moreover, PKGI.alpha. protein expression was dramatically increased in the lumbar spinal cord after noxious stimulation. This upregulation of PKGI.alpha. expression was completely blocked not only by a neuronal NO synthase inhibitor, and a sol. guanylate cyclase inhibitor, but also by an N-methyl-D-aspartate (***NMDA***) receptor antagonist, MK-801. Noxious stimulation not only initially activates but also later upregulates PKGI.alpha. expression in the superficial laminae via an ***NMDA*** -NO-cGMP signaling pathway, suggesting that PKGI.alpha. plays an important role in the central mechanism of inflammatory hyperalgesia in the spinal cord.

REFERENCE COUNT: 62 THERE ARE 62 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1997:222553 CAPLUS
DOCUMENT NUMBER: 126:259055
TITLE: Inhalational anesthetic effects on rat cerebellar nitric oxide and cyclic guanosine monophosphate production
AUTHOR(S): Rengasamy, Appavoo; Pajewski, Thomas N.; ***Johns,*** ***
Roger A.***
CORPORATE SOURCE: Department of Anesthesiology, University of Virginia Health Sciences Center, Charlottesville, VA, 22908, USA
SOURCE: Anesthesiology (1997), 86(3), 689-698
CODEN: ANESAV; ISSN: 0003-3022
PUBLISHER: Lippincott-Raven
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Inhalational anesthetics interact with the nitric oxide-cyclic guanosine monophosphate (NO-cGMP) pathway in the central nervous system (CNS) and attenuate excitatory neurotransmitter-induced cGMP concn. The site of anesthetic action on the NO-cGMP pathway in the CNS remains controversial. This study investigated the effect of inhalational anesthetics on N-methyl-D-aspartate (***NMDA***)-stimulated NO synthase activity and cGMP prodn. in rat cerebellum slices. The interaction of inhalational anesthetics with NO synthase activation and cGMP concn. was detd. in cerebellum slices of 10-day-old rats. Nitric oxide synthase activity in cerebellum slices was assessed by measuring the conversion of L-[3H]arginine to L-[3H]citrulline. The cGMP content of cerebellum slices was measured by RIA. Isoflurane at 1.5% and 3% enhanced the ***NMDA*** -stimulated NO synthase activity by two times while halothane at 1.5% and 3% produced no significant effect. However, the ***NMDA*** -stimulated cGMP prodn. was inhibited by both anesthetic agents. The anesthetic inhibition of cGMP accumulation was not significantly altered by a mixt. of superoxide dismutase and catalase or by glycine, a coagonist of the ***NMDA*** receptor. The enhancement of ***NMDA*** -induced NO synthase activity by isoflurane and the inhibition of ***NMDA*** -stimulated cGMP prodn. by halothane and isoflurane suggests that

This inhibitory effect of anesthetics on cGMP accumulation is not due to either their interaction with the glycine binding site of the ***NMDA*** receptor or to the action of superoxide anions.

L16 ANSWER 6 OF 11 USPATFULL
ACCESSION NUMBER: 2003:30917 USPATFULL
TITLE: Isoform specific inhibition for treatment of pain and reduction of anesthetic threshold
INVENTOR(S): Tao, Yuanxiang, Baltimore, MD, UNITED STATES
Johns, Roger A. , Reistertown, MD, UNITED STATES
PATENT ASSIGNEE(S): The Johns Hopkins University, Baltimore, MD (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003022866	A1	20030130
APPLICATION INFO.:	US 2002-183635	A1	20020628 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 2000-731876, filed on 8 Dec 2000, GRANTED, Pat. No. US 6476007

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-170260P	19991208 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 46
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 1009
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Several lines of evidence have shown a role for the nitric oxide (NO)/cyclic guanosine monophosphate (cGMP) signaling pathway in the development of spinal hyperalgesia. However, the roles of effectors for cGMP are not fully understood in the processing of pain in the spinal cord. cGMP-dependent protein kinase (PKG) I.alpha. but not PKGI.beta. was localized in the neuronal bodies and processes, and was distributed primarily in the superficial laminae of the spinal cord. Intrathecal administration of an inhibitor of PKGI.alpha., Rp-8-[(4-Chlorophenyl)thio]-cGMPS triethylamine, produces significant antinociception. Moreover, PKGI.alpha. protein expression was dramatically increased in the lumbar spinal cord after noxious stimulation. This upregulation of PKGI.alpha. expression was completely blocked not only by a neuronal NO synthase inhibitor, and a soluble guanylate cyclase inhibitor, but also by an N-methyl-D-aspartate (***NMDA***) receptor antagonist, MK-801. Noxious stimulation not only initially activates but also later upregulates PKGI.alpha. expression in the superficial laminae via an ***NMDA*** -NO-cGMP signaling pathway, suggesting that PKGI.alpha. plays an important role in the central mechanism of inflammatory hyperalgesia in the spinal cord.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 7 OF 11 USPATFULL
ACCESSION NUMBER: 2002:85548 USPATFULL
TITLE: Inhibition of interaction of PSD93 and PSD95 with nNOS and ***NMDA*** receptors
INVENTOR(S): Tao, Yuanxiang, Baltimore, MD, UNITED STATES
Johns, Roger A. , Reistertown, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002045590	A1	20020418
APPLICATION INFO.:	US 2001-853895	A1	20010514 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-242580P	20001023 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 65

NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 1513
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB PSD-95/SAP90 antisense-treated animals not only experience a significant decrease in MAC for isoflurane, but also experience an attenuation in the ***NMDA*** -induced increase in isoflurane MAC. PSD-95/SAP90 appears to mediate the role of the ***NMDA*** receptor in determining the MAC of inhalational anesthetics. Suppression of the expression of PSD-95/SAP90 in the spinal cord significantly attenuates responses to painful stimuli mediated through the N-methyl-D-aspartate receptor activation. In spinal cord neurons PSD-95/SAP90 interacts with the N-methyl-D-aspartate receptor subunits 2A/2B. Activation of the N-methyl-D-aspartate receptor in spinal hyperalgesia results in association of the N-methyl-D-aspartate receptor with PSD-95/SAP90. PSD-95/SAP90 is required for hyperalgesia triggered via the N-methyl-D-aspartate receptor at the spinal cord level.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 8 OF 11 USPATFULL
ACCESSION NUMBER: 2001:182591 USPATFULL
TITLE: Isoform specific inhibition for treatment of pain and reduction of anesthetic threshold
INVENTOR(S): Tao, Yuanxiang, Baltimore, MD, United States
Johns, Roger A. , Reistertown, MD, United States

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2001031750	A1	20011018	
	US 6476007	B2	20021105	
APPLICATION INFO.:	US 2000-731876	A1	20001208	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-170260P	19991208 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 46
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 1010
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Several lines of evidence have shown a role for the nitric oxide (NO)/cyclic guanosine monophosphate (cGMP) signaling pathway in the development of spinal hyperalgesia. However, the roles of effectors for cGMP are not fully understood in the processing of pain in the spinal cord. cGMP-dependent protein kinase (PKG) I.alpha. but not PKGI.beta. was localized in the neuronal bodies and processes, and was distributed primarily in the superficial laminae of the spinal cord. Intrathecal administration of an inhibitor of PKGI.alpha., Rp-8-[(4-Chlorophenyl)thio]-cGMPS triethylamine, produces significant antinociception. Moreover, PKGI.alpha. protein expression was dramatically increased in the lumbar spinal cord after noxious stimulation. This upregulation of PKGI.alpha. expression was completely blocked not only by a neuronal NO synthase inhibitor, and a soluble guanylate cyclase inhibitor, but also by an N-methyl-D-aspartate (***NMDA***) receptor antagonist, MK-801. Noxious stimulation not only initially activates but also later upregulates PKGI.alpha. expression in the superficial laminae via an ***NMDA*** -NO-cGMP signaling pathway, suggesting that PKGI.alpha. plays an important role in the central mechanism of inflammatory hyperalgesia in the spinal cord.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 9 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2003:90165 BIOSIS
DOCUMENT NUMBER: PREV200300090165
TITLE: Evidence of the Involvement of cGMP-Dependent Protein Kinase I alpha in Spinal Processing of Nociceptive Information.
AUTHOR(S): Tao, Yuan-Xiang (1); ***Johns, Roger A. (1)*** ; Hassan, Aalya (1); Haddad, Elie (1)
CORPORATE SOURCE: (1) Department of Anesthesiology and Critical Care

Baltimore, MD, USA USA
SOURCE: Anesthesiology Abstracts of Scientific Papers Annual Meeting, (2002) No. 2000, pp. Abstract No. 972. http://www.asa-abstracts.com. cd-rom. Meeting Info.: 2000 Annual Meeting of the American Society of Anesthesiologists San Francisco, CA, USA October 16-18, 2000 American Society of Anesthesiologists Inc.
.
DOCUMENT TYPE: Conference
LANGUAGE: English
AB Nitric oxide (NO) - cyclic guanosine 3', 5'-monophosphate (cGMP) signaling pathway is present in the neurons of the spinal cord and contributes to the development of hyperalgesia. Noxious stimulation increased NO synthase expression and cGMP content in spinal dorsal horn. NO donors and cGMP analogs applied intrathecally resulted in thermal hyperalgesia. Administration of inhibitors of NO synthase and soluble guanylate cyclase caused antinociception. cGMP-dependent protein kinases (PKGs) serve as major effectors for NO-cGMP signaling pathway in the nervous system. The prominent function for NO-cGMP signaling pathway in spinal hyperalgesia led us to hypothesize the possible roles for PKG isoforms in this response. In the present study, we first observed whether two isoforms of PKGI, I alpha and I beta, were expressed in the spinal cord. Second, we tested whether PKGIalpha contributed to spinal hyperalgesia produced by formalin and to formalin-induced c-fos expression as a marker of functional activity of nociceptive neurons in spinal cord. Third, we investigated whether activation of PKGIalpha is required for N-methyl-D-aspartate (***NMDA***)- or NO-produced spinal thermal hyperalgesia. For immunocytochemistry, the rats were perfused with 4% paraformaldehyde. The whole spinal cord was removed and frozen-sectioned at 30 mum. Sections were processed for immunocytochemistry with use of polyclonal rabbit anti-PKGIalpha, PKGIbeta and Fos antibodies. For behavioral testing, a PE-10 catheter was inserted into rat subarachnoid space through an incision in the atlanto-occipital membrane to a position 8-8.5 cm caudal to the cisterna. In the formalin test, three doses of a selective PKGIalpha inhibitor, Rp-8-p-CPT-cGMPS (10, 20, 30 mug /10 mul), were injected intrathecally 10 min prior to injection of 4% formalin (100 mul) into a hind paw. The pain-related behaviors, flinches and shakes, were assessed for 1h. In the tail-flick test, three doses of Rp-8-p-CPT-cGMPS were administrated 10 min prior to intrathecal injection of ***NMDA*** (10 nmol /10 mul) and NOC-12 (NO donor, 30 mug / 10 mul). Nociception was assessed by the time required to induce tail flick after applying radiant heat to the skin of the tail. PKGIalpha but not Ibeta was localized in the neuronal bodies and processes, and was distributed primarily in superficial dorsal horn. Intrathecal administration of Rp-8-p-CPT-cGMPS produced a significant antinociception demonstrated by the decrease in the number of flinches and shakes in the formalin test. This was accompanied by a marked reduction in formalin-induced c-fos expression in the spinal dorsal horn. ***NMDA*** - or NOC-12-produced facilitation of the tail-flick was significantly blocked by Rp-8-p-CPT-cGMPS. Rp-8-p-CPT-cGMPS given alone did not alter baseline tail-flick latency. Our results provide strong evidence that PKGIalpha is involved in spinal processing of nociceptive information.

L16 ANSWER 10 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2003:32053 BIOSIS
DOCUMENT NUMBER: PREV200300032053
TITLE: Isoform specific inhibition for treatment of pain and reduction of anesthetic threshold.
AUTHOR(S): Tao, Yuanxiang (1); ***Johns, Roger A.***
CORPORATE SOURCE: (1) Baltimore, MD, USA USA
ASSIGNEE: The Johns Hopkins University
PATENT INFORMATION: US 6476007 November 05, 2002
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Nov. 5 2002) Vol. 1264, No. 1, pp. No Pagination. http://www.uspto.gov/web/menu/patdata.html. e-file.
ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
AB Several lines of evidence have shown a role for the nitric oxide (NO)/cyclic guanosine monophosphate (cGMP) signaling pathway in the development of spinal hyperalgesia. However, the roles of effectors for cGMP are not fully understood in the processing of pain in the spinal cord. cGMP-dependent protein kinase (PKG) Ialpha but not PKGIbeta was localized in the neuronal bodies and processes, and was distributed primarily in the superficial laminae of the spinal cord. Intrathecal

cGMPS triethylamine, produces significant antinociception. Moreover, PKGIalpha protein expression was dramatically increased in the lumbar spinal cord after noxious stimulation. This upregulation of PKGIalpha expression was completely blocked not only by a neuronal NO synthase inhibitor, and a soluble guanylate cyclase inhibitor, but also by an N-methyl-D-aspartate (***NMDA***) receptor antagonist, MK-801. Noxious stimulation not only initially activates but also later upregulates PKGIalpha expression in the superficial laminae via an ***NMDA*** -NO-cGMP signaling pathway, suggesting that PKGIalpha plays an important role in the central mechanism of inflammatory hyperalgesia in the spinal cord.

L16 ANSWER 11 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:190563 BIOSIS
DOCUMENT NUMBER: PREV200000190563
TITLE: Activation of cGMP-dependent protein kinase Ialpha is required for N-methyl-D-aspartate- or nitric oxide-produced spinal thermal hyperalgesia.
AUTHOR(S): Tao, Yuan-Xiang; ***Johns, Roger A. (1)***
CORPORATE SOURCE: (1) Department of Anesthesiology and Critical Care Medicine, Johns Hopkins University School of Medicine, Blalock 1415, 600 North Wolfe Street, Baltimore, MD, 21287-4965 USA
SOURCE: European Journal of Pharmacology, (March 31, 2000) Vol. 392, No. 3, pp. 141-145.
ISSN: 0014-2999.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The effect of a selective cyclic guanocine 3',5'-monophosphate (cGMP)-dependent protein kinase Ialpha inhibitor, Rp-8-((4-chlorophenyl)thio)-cGMPS triethylamine (Rp-8-p-CPT-CGMPS), on either N-methyl-D-aspartate (***NMDA***)- or N-ethyl-2-(1-ethyl-2-hydroxy-2-nitrosohydrazino)ethanamine (NOC-12, a nitric oxide (NO) donor)-produced thermal hyperalgesia was examined in the rat. Intrathecal administration of ***NMDA*** (15 pg/10 mul) or NOC-12 (10, 20 and 30 mug/10 mul) produced a marked curtailment of the tail-flick latency. Maximal ***NMDA*** - or NOC-12-produced facilitation of the tail-flick reflex was significantly and dose-dependently blocked by intrathecal pretreatment with Rp-8-p-CPT-CGMPS (7.5, 15 and 30 mug/10 mul). Rp-8-p-CPT-CGMPS given alone did not markedly alter baseline tail-flick latency. These results suggest that the activation of cGMP-dependent protein kinase Ialpha is required for ***NMDA*** - or NO-produced facilitation of thermal hyperalgesia at the spinal cord level.

=> d history

(FILE 'HOME' ENTERED AT 17:12:53 ON 25 FEB 2003)

FILE 'MEDLINE, CAPLUS, LIFESCI, EMBASE, USPATFULL, BIOSIS' ENTERED AT 17:13:20 ON 25 FEB 2003
L1 2 S HALOETHANE AND PAIN
L2 67441 S HALOTHANE
L3 2608 S L2 AND PAIN
L4 475 S L3 AND INTRATHECAL?
L5 2 S L2 AND PSD93
L6 2 DUP REM L5 (0 DUPLICATES REMOVED)
L7 373 DUP REM L4 (102 DUPLICATES REMOVED)
L8 216 S L7 AND ANESTHESIA
L9 0 S HALOETHANE (P) INTRATHECAL?
L10 13 S PSD93 AND NMDA
L11 12 DUP REM L10 (1 DUPLICATE REMOVED)
L12 5 S ((PSD()93) OR (CHAPSYN()110)) AND (PAIN OR ANESTHE?)
L13 5 DUP REM L12 (0 DUPLICATES REMOVED)
E JOHNS R?/AU
E JOHNS ROGER?/AU
L14 164 S E1
L15 116 DUP REM L14 (48 DUPLICATES REMOVED)
L16 11 S L15 AND NMDA

=> s l15 and psd93
L17 2 L15 AND PSD93

=> d l17

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AN   2001:850924  CAPLUS
DN   135:366767
TI   Inhibition of interaction of   ***psd93***   and psd95 with neuronal
     nitric oxide synthase and NMDA receptors
IN     ***Johns, Roger A.***  ; Tao, Yuanxiang
PA   The Johns Hopkins University, USA
SO   PCT Int. Appl., 45 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.         KIND  DATE           APPLICATION NO.  DATE
     ---------------    ----  --------       ---------------  --------
PI   WO 2001087285      A2    20011122       WO 2001-US15372  20010514
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT,
             RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US,
             UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
        RW:  GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
             DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
             BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     US 2002045590      A1    20020418       US 2001-853895   20010514
PRAI US 2000-203894P    P     20000512
     US 2000-242580P    P     20001023


=> d l17 2

L17  ANSWER 2 OF 2  USPATFULL
AN     2002:85548  USPATFULL
TI     Inhibition of interaction of   ***PSD93***   and PSD95 with nNOS and
       NMDA receptors
IN     Tao, Yuanxiang, Baltimore, MD, UNITED STATES
         ***Johns, Roger A.***  , Reistertown, MD, UNITED STATES
PI     US 2002045590       A1   20020418
AI     US 2001-853895      A1   20010514 (9)
PRAI   US 2000-242580P      20001023 (60)
DT     Utility
FS     APPLICATION
LN.CNT 1513
INCL   INCLM: 514/044.000
NCL    NCLM:  514/044.000
IC     [7]
       ICM: A61K048-00
CAS INDEXING IS AVAILABLE FOR THIS PATENT.


=> s l15 and (psd()93)
L18           1 L15 AND (PSD(W) 93)

=> d l18

L18  ANSWER 1 OF 1  USPATFULL
AN     2002:85548  USPATFULL
TI     Inhibition of interaction of PSD93 and PSD95 with nNOS and NMDA
       receptors
IN     Tao, Yuanxiang, Baltimore, MD, UNITED STATES
         ***Johns, Roger A.***  , Reistertown, MD, UNITED STATES
PI     US 2002045590       A1   20020418
AI     US 2001-853895      A1   20010514 (9)
PRAI   US 2000-242580P      20001023 (60)
DT     Utility
FS     APPLICATION
LN.CNT 1513
INCL   INCLM: 514/044.000
NCL    NCLM:  514/044.000
IC     [7]
       ICM: A61K048-00
CAS INDEXING IS AVAILABLE FOR THIS PATENT.


=> e tao Y?/au
E1            3     TAO Y Y/AU
E2           15     TAO Y Z/AU
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E4            1     TAO YA JUN/AU
E5            2     TAO YA QIU/AU
E6            7     TAO YA XIONG/AU
E7            1     TAO YAJU/AU
E8            1     TAO YAMING/AU
E9           18     TAO YAN/AU
E10           5     TAO YAN CHUN/AU
E11           1     TAO YAN MEI/AU
E12           3     TAO YANCHUN/AU

=> e tao Yu?/au
E1            1     TAO YU XIANG/AU
E2            2     TAO YU YUN/AU
E3            0 --> TAO YU?/AU
E4            2     TAO YUAN/AU
E5            3     TAO YUAN BO/AU
E6            3     TAO YUAN KAI/AU
E7           17     TAO YUAN XIANG/AU
E8            1     TAO YUAN XIANG WEI FENG/AU
E9            1     TAO YUANGI/AU
E10          18     TAO YUANJIN/AU
E11           1     TAO YUANJUN/AU
E12          19     TAO YUANQI/AU

=> s e7 or e8
L19          18 "TAO YUAN XIANG"/AU OR "TAO YUAN XIANG WEI FENG"/AU

=> e tao Yuanx?/au
E1            1     TAO YUANJUN/AU
E2           19     TAO YUANQI/AU
E3            0 --> TAO YUANX?/AU
E4           14     TAO YUANXIANG/AU
E5            3     TAO YUANXIAO/AU
E6            2     TAO YUCHUN/AU
E7           17     TAO YUE/AU
E8            2     TAO YUE DUO/AU
E9            1     TAO YUE QUN/AU
E10           4     TAO YUE WU/AU
E11           3     TAO YUEDUO/AU
E12           3     TAO YUEFEI/AU

=> s e4
L20          14 "TAO YUANXIANG"/AU

=> s l19 or l20
L21          32 L19 OR L20

=> s l21 and nmda
L22          14 L21 AND NMDA

=> dup rem l22
PROCESSING COMPLETED FOR L22
L23          11 DUP REM L22 (3 DUPLICATES REMOVED)

=> d l23 ibib abs tot

L23  ANSWER 1 OF 11  USPATFULL
ACCESSION NUMBER:        2003:30917  USPATFULL
TITLE:                   Isoform specific inhibition for treatment of pain and
                         reduction of anesthetic threshold
INVENTOR(S):               ***Tao, Yuanxiang***  , Baltimore, MD, UNITED STATES
                         Johns, Roger A., Reistertown, MD, UNITED STATES
PATENT ASSIGNEE(S):      The Johns Hopkins University, Baltimore, MD (U.S.
                         corporation)

                              NUMBER        KIND    DATE
                         ---------------- ------ --------
PATENT INFORMATION:      US 2003022866      A1   20030130
APPLICATION INFO.:       US 2002-183635     A1   20020628  (10)
RELATED APPLN. INFO.:    Division of Ser. No. US 2000-731876, filed on 8 Dec
                         2000, GRANTED, Pat. No. US 6476007

                               NUMBER         DATE
                         ------------------ --------
PRIORITY INFORMATION:    US 1999-170260P    19991208 (60)
DOCUMENT TYPE:           Utility
```

LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 46
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 1009
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Several lines of evidence have shown a role for the nitric oxide (NO)/cyclic guanosine monophosphate (cGMP) signaling pathway in the development of spinal hyperalgesia. However, the roles of effectors for cGMP are not fully understood in the processing of pain in the spinal cord. cGMP-dependent protein kinase (PKG) I.alpha. but not PKGI.beta. was localized in the neuronal bodies and processes, and was distributed primarily in the superficial laminae of the spinal cord. Intrathecal administration of an inhibitor of PKGI.alpha., Rp-8-[(4-Chlorophenyl)thio]-cGMPS triethylamine, produces significant antinociception. Moreover, PKGI.alpha. protein expression was dramatically increased in the lumbar spinal cord after noxious stimulation. This upregulation of PKGI.alpha. expression was completely blocked not only by a neuronal NO synthase inhibitor, and a soluble guanylate cyclase inhibitor, but also by an N-methyl-D-aspartate (***NMDA***) receptor antagonist, MK-801. Noxious stimulation not only initially activates but also later upregulates PKGI.alpha. expression in the superficial laminae via an ***NMDA*** -NO-cGMP signaling pathway, suggesting that PKGI.alpha. plays an important role in the central mechanism of inflammatory hyperalgesia in the spinal cord.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L23 ANSWER 2 OF 11 USPATFULL
ACCESSION NUMBER: 2002:85548 USPATFULL
TITLE: Inhibition of interaction of PSD93 and PSD95 with nNOS and ***NMDA*** receptors
INVENTOR(S): ***Tao, Yuanxiang*** , Baltimore, MD, UNITED STATES
Johns, Roger A., Reistertown, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002045590	A1	20020418
APPLICATION INFO.:	US 2001-853895	A1	20010514 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-242580P	20001023 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 65
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 1513
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB PSD-95/SAP90 antisense-treated animals not only experience a significant decrease in MAC for isoflurane, but also experience an attenuation in the ***NMDA*** -induced increase in isoflurane MAC. PSD-95/SAP90 appears to mediate the role of the ***NMDA*** receptor in determining the MAC of inhalational anesthetics. Suppression of the expression of PSD-95/SAP90 in the spinal cord significantly attenuates responses to painful stimuli mediated through the N-methyl-D-aspartate receptor activation. In spinal cord neurons PSD-95/SAP90 interacts with the N-methyl-D-aspartate receptor subunits 2A/2B. Activation of the N-methyl-D-aspartate receptor in spinal hyperalgesia results in association of the N-methyl-D-aspartate receptor with PSD-95/SAP90. PSD-95/SAP90 is required for hyperalgesia triggered via the N-methyl-D-aspartate receptor at the spinal cord level.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L23 ANSWER 3 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2003:32053 BIOSIS
DOCUMENT NUMBER: PREV200300032053
TITLE: Isoform specific inhibition for treatment of pain and reduction of anesthetic threshold.
AUTHOR(S): ***Tao, Yuanxiang (1)*** ; Johns, Roger A.
CORPORATE SOURCE: (1) Baltimore, MD, USA USA

PATENT INFORMATION: US 6476007 November 05, 2002
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Nov. 5 2002) Vol. 1264, No. 1, pp. No Pagination. http://www.uspto.gov/web/menu/patdata.html. e-file.
ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
AB Several lines of evidence have shown a role for the nitric oxide (NO)/cyclic guanosine monophosphate (cGMP) signaling pathway in the development of spinal hyperalgesia. However, the roles of effectors for cGMP are not fully understood in the processing of pain in the spinal cord. cGMP-dependent protein kinase (PKG) Ialpha but not PKGIbeta was localized in the neuronal bodies and processes, and was distributed primarily in the superficial laminae of the spinal cord. Intrathecal administration of an inhibitor of PKGIalpha, Rp-8-[(4-Chlorophenyl)thio]-cGMPS triethylamine, produces significant antinociception. Moreover, PKGIalpha protein expression was dramatically increased in the lumbar spinal cord after noxious stimulation. This upregulation of PKGIalpha expression was completely blocked not only by a neuronal NO synthase inhibitor, and a soluble guanylate cyclase inhibitor, but also by an N-methyl-D-aspartate (***NMDA***) receptor antagonist, MK-801. Noxious stimulation not only initially activates but also later upregulates PKGIalpha expression in the superficial laminae via an ***NMDA*** -NO-cGMP signaling pathway, suggesting that PKGIalpha plays an important role in the central mechanism of inflammatory hyperalgesia in the spinal cord.

L23 ANSWER 4 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2003:90165 BIOSIS
DOCUMENT NUMBER: PREV200300090165
TITLE: Evidence of the Involvement of cGMP-Dependent Protein Kinase I alpha in Spinal Processing of Nociceptive Information.
AUTHOR(S): ***Tao, Yuan-Xiang (1)*** ; Johns, Roger A. (1); Hassan, Aalya (1); Haddad, Elie (1)
CORPORATE SOURCE: (1) Department of Anesthesiology and Critical Care Medicine, Johns Hopkins University School of Medicine, Baltimore, MD, USA USA
SOURCE: Anesthesiology Abstracts of Scientific Papers Annual Meeting, (2002) No. 2000, pp. Abstract No. 972. http://www.asa-abstracts.com. cd-rom.
Meeting Info.: 2000 Annual Meeting of the American Society of Anesthesiologists San Francisco, CA, USA October 16-18, 2000 American Society of Anesthesiologists Inc.
.
DOCUMENT TYPE: Conference
LANGUAGE: English
AB Nitric oxide (NO) - cyclic guanosine 3', 5'-monophosphate (cGMP) signaling pathway is present in the neurons of the spinal cord and contributes to the development of hyperalgesia. Noxious stimulation increased NO synthase expression and cGMP content in spinal dorsal horn. NO donors and cGMP analogs applied intrathecally resulted in thermal hyperalgesia. Administration of inhibitors of NO synthase and soluble guanylate cyclase caused antinociception. cGMP-dependent protein kinases (PKGs) serve as major effectors for NO-cGMP signaling pathway in the nervous system. The prominent function for NO-cGMP signaling pathway in spinal hyperalgesia led us to hypothesize the possible roles for PKG isoforms in this response. In the present study, we first observed whether two isoforms of PKGI, I alpha and I beta, were expressed in the spinal cord. Second, we tested whether PKGIalpha contributed to spinal hyperalgesia produced by formalin and to formalin-induced c-fos expression as a marker of functional activity of nociceptive neurons in spinal cord. Third, we investigated whether activation of PKGIalpha is required for N-methyl-D-aspartate (***NMDA***)- or NO-produced spinal thermal hyperalgesia. For immunocytochemistry, the rats were perfused with 4% paraformaldehyde. The whole spinal cord was removed and frozen-sectioned at 30 mum. Sections were processed for immunocytochemistry with use of polyclonal rabbit anti-PKGIalpha, PKGIbeta and Fos antibodies. For behavioral testing, a PE-10 catheter was inserted into rat subarachnoid space through an incision in the atlanto-occipital membrane to a position 8-8.5 cm caudal to the cisterna. In the formalin test, three doses of a selective PKGIalpha inhibitor, Rp-8-p-CPT-cGMPS (10, 20, 30 mug /10 mul), were injected intrathecally 10 min prior to injection of 4% formalin (100 mul) into a hind paw. The pain-related behaviors, flinches and shakes, were assessed for 1h. In the tail-flick test, three doses of

of ***NMDA*** (10 nmol /10 mul) and NOC-12 (NO donor, 30 mug / 10 mul). Nociception was assessed by the time required to induce tail flick after applying radiant heat to the skin of the tail. PKGIalpha but not Ibeta was localized in the neuronal bodies and processes, and was distributed primarily in superficial dorsal horn. Intrathecal administration of Rp-8-p-CPT-cGMPS produced a significant antinociception demonstrated by the decrease in the number of flinches and shakes in the formalin test. This was accompanied by a marked reduction in formalin-induced c-fos expression in the spinal dorsal horn. ***NMDA*** - or NOC-12-produced facilitation of the tail-flick was significantly blocked by Rp-8-p-CPT-cGMPS. Rp-8-p-CPT-cGMPS given alone did not alter baseline tail-flick latency. Our results provide strong evidence that PKGIalpha is involved in spinal processing of nociceptive information.

L23 ANSWER 5 OF 11 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:850924 CAPLUS
DOCUMENT NUMBER: 135:366767
TITLE: Inhibition of interaction of psd93 and psd95 with neuronal nitric oxide synthase and ***NMDA*** receptors
INVENTOR(S): Johns, Roger A.; ***Tao, Yuanxiang***
PATENT ASSIGNEE(S): The Johns Hopkins University, USA
SOURCE: PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001087285	A2	20011122	WO 2001-US15372	20010514
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2002045590	A1	20020418	US 2001-853895	20010514

PRIORITY APPLN. INFO.: US 2000-203894P P 20000512
US 2000-242580P P 20001023

AB PSD-95/SAP90 antisense-treated animals not only experience a significant decrease in min. alveolar concn. (MAC) for isoflurane, but also experience an attenuation in the ***NMDA*** -induced increase in isoflurane MAC. PSD-95/SAP90 appears to mediate the role of the ***NMDA*** receptor in detg. the MAC of inhalational anesthetics. Suppression of the expression of PSD-95/SAP90 in the spinal cord significantly attenuates responses to painful stimuli mediated through the N-methyl-D-aspartate receptor activation. In spinal cord neurons PSD-95/SAP90 interacts with the N-methyl-D-aspartate receptor subunits 2A/2B. Activation of the N-methyl-D-aspartate receptor in spinal hyperalgesia results in assocn. of the N-methyl-D-aspartate receptor with PSD-95/SAP90. PSD-95/SAP90 is required for hyperalgesia triggered via the N-methyl-D-aspartate receptor at the spinal cord level.

L23 ANSWER 6 OF 11 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:434853 CAPLUS
DOCUMENT NUMBER: 135:29155
TITLE: Cyclic GMP-dependent protein kinase isoform-specific inhibition for treatment of pain and reduction of anesthetic threshold
INVENTOR(S): Johns, Roger A.; ***Tao, Yuanxiang***
PATENT ASSIGNEE(S): The Johns Hopkins University, USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001041752	A2	20010614	WO 2000-US33195	20001208

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 2001031750	A1	20011018	US 2000-731876	20001208
US 6476007	B2	20021105		
US 2003022866	A1	20030130	US 2002-183635	20020628

PRIORITY APPLN. INFO.: US 1999-170260P A1 19991208
US 2000-731876 A3 20001208

AB Several lines of evidence have shown a role for the nitric oxide (NO)/cyclic guanosine monophosphate (cGMP) signaling pathway in the development of spinal hyperalgesia. However, the roles of effectors for cGMP are not fully understood in the processing of pain in the spinal cord. CGMP-dependent protein kinase (PKG) I.alpha. but not PKGI.beta. was localized in the neuronal bodies and processes, and was distributed primarily in the superficial laminae of the spinal cord. Intrathecal administration of an inhibitor of PKGI.alpha., Rp-8-[(4-Chlorophenyl)thio]-cGMPS triethylamine, produces significant antinociception. Moreover, PKGI.alpha. protein expression was dramatically increased in the lumbar spinal cord after noxious stimulation. This upregulation of PKGI.alpha. expression was completely blocked not only by a neuronal NO synthase inhibitor, and a sol. guanylate cyclase inhibitor, but also by an N-methyl-D-aspartate (***NMDA***) receptor antagonist, MK-801. Noxious stimulation not only initially activates but also later upregulates PKGI.alpha. expression in the superficial laminae via an ***NMDA*** -NO-cGMP signaling pathway, suggesting that PKGI.alpha. plays an important role in the central mechanism of inflammatory hyperalgesia in the spinal cord.

REFERENCE COUNT: 62 THERE ARE 62 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 7 OF 11 USPATFULL

ACCESSION NUMBER: 2001:182591 USPATFULL

TITLE: Isoform specific inhibition for treatment of pain and reduction of anesthetic threshold

INVENTOR(S): ***Tao, Yuanxiang*** , Baltimore, MD, United States
Johns, Roger A., Reistertown, MD, United States

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2001031750	A1	20011018	
	US 6476007	B2	20021105	
APPLICATION INFO.:	US 2000-731876	A1	20001208	(9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-170260P	19991208 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 46
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 1010
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Several lines of evidence have shown a role for the nitric oxide (NO)/cyclic guanosine monophosphate (cGMP) signaling pathway in the development of spinal hyperalgesia. However, the roles of effectors for cGMP are not fully understood in the processing of pain in the spinal cord. cGMP-dependent protein kinase (PKG) I.alpha. but not PKGI.beta. was localized in the neuronal bodies and processes, and was distributed primarily in the superficial laminae of the spinal cord. Intrathecal administration of an inhibitor of PKGI.alpha., Rp-8-[(4-Chlorophenyl)thio]-cGMPS triethylamine, produces significant antinociception. Moreover, PKGI.alpha. protein expression was dramatically increased in the lumbar spinal cord after noxious stimulation. This upregulation of PKGI.alpha. expression was completely blocked not only by a neuronal NO synthase inhibitor, and a soluble guanylate cyclase inhibitor, but also by an N-methyl-D-aspartate (***NMDA***) receptor antagonist, MK-801. Noxious stimulation not only

the superficial laminae via an ***NMDA*** -NO-cGMP signaling pathway, suggesting that PKGI.alpha. plays an important role in the central mechanism of inflammatory hyperalgesia in the spinal cord.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L23 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 1
ACCESSION NUMBER: 2001:839105 CAPLUS
DOCUMENT NUMBER: 136:353639
TITLE: Knockdown of PSD-95/SAP90 delays the development of neuropathic pain in rats
AUTHOR(S): Tao, Feng; ***Tao, Yuan-Xiang*** ; Gonzalez, Julio A.; Fang, Ming; Mao, Peizhong; Johns, Roger A.
CORPORATE SOURCE: Department of Anesthesiology and Critical Care Medicine, Johns Hopkins University School of Medicine, Baltimore, MD, 21287-4965, USA
SOURCE: NeuroReport (2001), 12(15), 3251-3255
CODEN: NERPEZ; ISSN: 0959-4965
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Our previous work has shown that PSD-95/SAP90 is required for ***NMDA*** receptor-mediated thermal hyperalgesia. To address the role of PSD-95/SAP90 in chronic pain, the present study investigated the effect of the deficiency of PSD-95/SAP90 on nerve injury-induced neuropathic pain. Following unilateral L5 spinal nerve injury, mech. and thermal hyperalgesia developed within 3 days and persisted for 9 days or longer on the injured side. The intrathecal administration of antisense oligodeoxynucleotide specifically against PSD-95/SAP90, but not sense or missense oligodeoxynucleotide, dose-dependently delayed the onset of tactile allodynia and thermal hyperalgesia. These results suggest that PSD-95/SAP90 might be involved in the central mechanisms of the development of chronic neuropathic pain.
REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 2
ACCESSION NUMBER: 2001:475912 CAPLUS
DOCUMENT NUMBER: 136:210425
TITLE: Effect of the deficiency of spinal PSD-95/SAP90 on the minimum alveolar anesthetic concentration of isoflurane in rats
AUTHOR(S): ***Tao, Yuan-Xiang*** ; Johns, Roger A.
CORPORATE SOURCE: Department of Anesthesiology and Critical Care Medicine, Johns Hopkins University School of Medicine, Baltimore, MD, 21287-4965, USA
SOURCE: Anesthesiology (2001), 94(6), 1010-1015
CODEN: ANESAV; ISSN: 0003-3022
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Spinal N-methyl-D-aspartate (***NMDA***) receptor activation was demonstrated to play an important role in the processing of spinal nociceptive information and in the detn. of the min. alveolar anesthetic concn. (MAC) of inhalational anesthetics. Postsynaptic d.-95 (PSD-95)/synapse-assocd. protein-90 (SAP90), a mol. scaffolding protein that binds and clusters the ***NMDA*** receptor preferentially at synapses, was implicated in ***NMDA*** -induced thermal hyperalgesia. The current study investigated the possible involvement of PSD-95/SAP90 in detg. MAC for isoflurane anesthesia. Sprague-Dawley rats were pretreated intrathecally with PSD-95/SAP90 antisense oligodeoxyribonucleotide (ODN), sense ODN, missense ODN, or saline every 24 h for 4 days. After initial baseline detn. of the MAC, ***NMDA*** or saline was injected intrathecally. Ten minutes later, MAC measurement was repeated. The rats also were evaluated for the presence of locomotor dysfunction by intrathecal administration of ***NMDA*** or saline in the saline- and ODN-treated rats. In the groups treated with antisense ODNs, but not in those treated with sense or missense ODNs, there was a significant decrease in isoflurane MAC that was not accompanied by marked changes in either blood pressure or heart rate. In the saline-treated group, intrathecal ***NMDA*** caused an increase in isoflurane MAC. In contrast, in the antisense ODN-treated group, intrathecal ***NMDA*** did not produce a significant change in isoflurane MAC. An ***NMDA*** -induced increase in blood pressure but not heart rate was found in both saline- and antisense ODN-treated groups. Locomotor activity was not changed in any of the treated animals. The results indicate not only a

NMDA -induced increase in isoflurane MAC in the PSD-95/SAP90 antisense-treated animals, which suggests that PSD-95/SAP90 may mediate the role of the ***NMDA*** receptor in detg. the MAC of inhalational anesthetics.
REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 10 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:190563 BIOSIS
DOCUMENT NUMBER: PREV200000190563
TITLE: Activation of cGMP-dependent protein kinase Ialpha is required for N-methyl-D-aspartate- or nitric oxide-produced spinal thermal hyperalgesia.
AUTHOR(S): ***Tao, Yuan-Xiang*** ; Johns, Roger A. (1)
CORPORATE SOURCE: (1) Department of Anesthesiology and Critical Care Medicine, Johns Hopkins University School of Medicine, Blalock 1415, 600 North Wolfe Street, Baltimore, MD, 21287-4965 USA
SOURCE: European Journal of Pharmacology, (March 31, 2000) Vol. 392, No. 3, pp. 141-145.
ISSN: 0014-2999.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The effect of a selective cyclic guanocine 3',5'-monophosphate (cGMP)-dependent protein kinase Ialpha inhibitor, Rp-8-((4-chlorophenyl)thio)-cGMPS triethylamine (Rp-8-p-CPT-CGMPS), on either N-methyl-D-aspartate (***NMDA***)- or N-ethyl-2-(1-ethyl-2-hydroxy-2-nitrosohydrazino)ethanamine (NOC-12, a nitric oxide (NO) donor)-produced thermal hyperalgesia was examined in the rat. Intrathecal administration of ***NMDA*** (15 pg/10 mul) or NOC-12 (10, 20 and 30 mug/10 mul) produced a marked curtailment of the tail-flick latency. Maximal ***NMDA*** - or NOC-12-produced facilitation of the tail-flick reflex was significantly and dose-dependently blocked by intrathecal pretreatment with Rp-8-p-CPT-CGMPS (7.5, 15 and 30 mug/10 mul). Rp-8-p-CPT-CGMPS given alone did not markedly alter baseline tail-flick latency. These results suggest that the activation of cGMP-dependent protein kinase Ialpha is required for ***NMDA*** - or NO-produced facilitation of thermal hyperalgesia at the spinal cord level.

L23 ANSWER 11 OF 11 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 3
ACCESSION NUMBER: 1998:742974 CAPLUS
DOCUMENT NUMBER: 130:106222
TITLE: ***NMDA*** receptors mediating Fos expression in rat spinal cord induced by subcutaneous injection of formalin
AUTHOR(S): ***Tao, Yuan-Xiang*** ; Zhao, Zhi-Qi
CORPORATE SOURCE: Shanghai Brain Research Institute, Chinese Academy of Sciences, Shanghai, 200031, Peop. Rep. China
SOURCE: Zhongguo Yaoli Xuebao (1998), 19(6), 506-509
CODEN: CYLPDN; ISSN: 0253-9756
PUBLISHER: Kexue Chubanshe
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Aim: To examine the effects of N-methyl-D-aspartate (***NMDA***) and non- ***NMDA*** receptors on noxious stimulation-induced Fos expression in the rat spinal cord. Methods: Formalin (2%) was injected s.c. into one hind-paw of the rat. Results: Two hours after s.c. formalin, Fos-like immunoreactive (FLI) neurons were distributed mainly in medial part of the lamina I and the outer lamina II of the ipsilateral dorsal horn. dl-2-Amino-5-phosphonovalerate administered intrathecally (10 .mu.L, 0.01, 0.1, or 1 g/L) before injection of formalin into a hind-paw reduced the no. of FLI neurons dose-dependently in the dorsal horn ($P<0.01$), while 6,7-dinitroquinoxaline-2,3(1H,4H)-dione (1 g/L) was ineffective. Conclusion: ***NMDA*** receptor mediated noxious stimulation-induced Fos expression in the rat spinal cord.
REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s l21 and psd93
L24 2 L21 AND PSD93

=> dup rem l24
PROCESSING COMPLETED FOR L24
L25 2 DUP REM L24 (0 DUPLICATES REMOVED)

=> d l25 ibib abs tot

L25 ANSWER 1 OF 2 USPATFULL
ACCESSION NUMBER: 2002:85548 USPATFULL
TITLE: Inhibition of interaction of ***PSD93*** and PSD95 with nNOS and NMDA receptors
INVENTOR(S): ***Tao, Yuanxiang*** , Baltimore, MD, UNITED STATES
Johns, Roger A., Reistertown, MD, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2002045590	A1	20020418	
APPLICATION INFO.:	US 2001-853895	A1	20010514	(9)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2000-242580P	20001023	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: BANNER & WITCOFF, 1001 G STREET N W, SUITE 1100, WASHINGTON, DC, 20001
NUMBER OF CLAIMS: 65
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 1513
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB PSD-95/SAP90 antisense-treated animals not only experience a significant decrease in MAC for isoflurane, but also experience an attenuation in the NMDA-induced increase in isoflurane MAC. PSD-95/SAP90 appears to mediate the role of the NMDA receptor in determining the MAC of inhalational anesthetics. Suppression of the expression of PSD-95/SAP90 in the spinal cord significantly attenuates responses to painful stimuli mediated through the N-methyl-D-aspartate receptor activation. In spinal cord neurons PSD-95/SAP90 interacts with the N-methyl-D-aspartate receptor subunits 2A/2B. Activation of the N-methyl-D-aspartate receptor in spinal hyperalgesia results in association of the N-methyl-D-aspartate receptor with PSD-95/SAP90. PSD-95/SAP90 is required for hyperalgesia triggered via the N-methyl-D-aspartate receptor at the spinal cord level.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L25 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:850924 CAPLUS
DOCUMENT NUMBER: 135:366767
TITLE: Inhibition of interaction of ***psd93*** and psd95 with neuronal nitric oxide synthase and NMDA receptors
INVENTOR(S): Johns, Roger A.; ***Tao, Yuanxiang***
PATENT ASSIGNEE(S): The Johns Hopkins University, USA
SOURCE: PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001087285	A2	20011122	WO 2001-US15372	20010514
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2002045590	A1	20020418	US 2001-853895	20010514

PRIORITY APPLN. INFO.: US 2000-203894P P 20000512
US 2000-242580P P 20001023

AB PSD-95/SAP90 antisense-treated animals not only experience a significant decrease in min. alveolar concn. (MAC) for isoflurane, but also experience an attenuation in the NMDA-induced increase in isoflurane MAC. PSD-95/SAP90 appears to mediate the role of the NMDA receptor in detg. the MAC of inhalational anesthetics. Suppression of the expression of

painful stimuli mediated through the N-methyl-D-aspartate receptor activation. In spinal cord neurons PSD-95/SAP90 interacts with the N-methyl-D-aspartate receptor subunits 2A/2B. Activation of the N-methyl-D-aspartate receptor in spinal hyperalgesia results in assocn. of the N-methyl-D-aspartate receptor with PSD-95/SAP90. PSD-95/SAP90 is required for hyperalgesia triggered via the N-methyl-D-aspartate receptor at the spinal cord level.

=>

---Logging off of STN---

=>
Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	266.67	266.88

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-13.02	-13.02

STN INTERNATIONAL LOGOFF AT 17:57:38 ON 25 FEB 2003

halothane

<chemical> A nonflammable, halogenated, hydrocarbon anaesthetic that provides relatively rapid induction with little or no excitement. Analgesia may not be adequate. Nitrous oxide is often given concomitantly. Because halothane may not produce sufficient muscle relaxation, supplemental neuromuscular blocking agents may be required.

Pharmacological action: anaesthetics, inhalation.

Chemical name: Ethane, 2-bromo-2-chloro-1,1,1-trifluoro-

(12 Dec 1998)

Previous: halorhodopsin, haloscope, halo sign, halo sign of hydrops, halosteresis
Next: halothane effect, halothane-ether azeotrope, halothane hepatitis